ENVIRONMENTAL HEALTH RISK ASSESSMENT OF PARTICULATE AIR POLLUTION AND MORTALITY IN METROPOLITAN MANILA, PHILIPPINES

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ABSTRACT

Key Words: Metropolitan Manila, Environmental Health Risk Assessment, Life Expectancy, Age, Educational Level, Particulate Air Pollution, PM_{10}

Background and Objectives: Metropolitan Manila is considered a Mega-City with approximately 10 million people as of 1995. Due to rapid industrialization and urbanization, environmental health problems including air pollution have become very prominent. In this study, the potential magnitude of environmental health inequalities with particular reference to particulate air pollution in Metropolitan Manila, Philippines has been assessed in terms of mortality by adapting the risk assessment method. Utilizing various indicators of mortality such as life expectancy and years of life lost, modification of the chronic health impact by markers of population heterogeneity particularly age and educational attainment has been explored In addition, the impact of various pollution reduction scenarios were evaluated.

Methodology: Published Environmental Health Risk Assessment methods were adapted and applied for estimating chronic mortality effects of PM_{10} pollution in the city of Metropolitan Manila. Pooled estimates derived from the exposure-response coefficients of the two US longitudinal epidemiological studies on PM_{10} and chronic mortality were used primarily. These pooled estimates which signify increases in mortality with incremental increase in PM10 were applied to the indicators of mortality. Hence, the methodology involved a life table approach using age-specific mortality rates from Metropolitan Manila in 1995. Annual averages of PM_{10} for the whole of Metropolitan Manila and for the cities within were also used. Life expectancies using two pollution reduction scenarios were compared with the 1995 life table to determine pollution reduction benefits. All causes and cardio-respiratory causes of deaths were evaluated. Apart from gains in life expectancy, other effect measures such as years of life loss and number of deaths were also assessed.

Findings: Health impact as a result of particulate pollution reduction by 10 $\mu g/m^3$ resulted in gains in life expectancies of approximately five months for both males

and females. Inclusion of effects on the elderly in the model did not make much of a difference in terms of life expectancy gains. However, with the addition of the effects on infants in the model and retaining the effects on adults and the elderly, life expectancy gains, years of life lost and attributable deaths increased. Life expectancy gains were also estimated to be more for the low education level as compared to the middle and high education levels. The overall life expectancy gains for a reduction to the international annual guideline of 50 $\mu g/m^3$ PM_{10} scenario were 2.22 years for males and 1.88 years for females. By educational level, the life expectancy gains at age 25 years old in the same pollution reduction scenario, range from 0.74 years for males and 0.59 years for females in the high educational level to more than four years for males and more than three and half years for females in the low educational level. Improvements in the cardio-respiratory causes of death alone contributed most to the life expectancy gains. The estimates that resulted from this assessment were found to be sensitive to the exposure-response coefficients used, the exposure reduction scenarios, the measures of heterogeneity (particularly age and educational level), baseline rates and the time period of effect. In addition, geographic differences in gains in life expectancy within Metropolitan Manila were like wise assessed. Higher gains in life expectancy were seen in the cities in the north where pollution levels are also greater than in the cities in the south.

Implication: The air pollution studies have found relatively small exposure response coefficients. However, the impact on public health is quite substantial and relevant to prioritising intervention to control air pollution. The results in this study could be used in several aspects of public policy as discussed in the thesis. These results were presented to decision-makers in the government and the responses summarised.

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INTRODUCTION

The life and health of a person is shaped by situations and changes in his/her physical environment, social situations, occupational exposures, emotional relationships, and generally, lifestyle. There are many interacting factors and arguably the urban life is more complex. The starting point of this project is a concern that such factors interact in these complex ways (*N. Krieger, 1994*) and that this complexity may be more revealing. In this project, it was decided to explore such complexity and interaction in the context of one aspect of environment and health: the health impact of air pollution and the modification of effects of air pollution by a number of markers of population heterogeneity.

This study aims to assess the potential magnitude of environmental health inequalities in a Mega-city in a developing country with particular reference to particulate air pollution in Metropolitan Manila. Present methods of Environmental Health Risk Assessment for air pollution can be applied in the city of Metropolitan Manila but would benefit from certain modifications, for example, with regards to the inclusion of age and socio-economic variables. The review is structured in such a way that relevant evidence in support of this approach is systematically presented. The review starts with a short background on urbanisation and health as well as the general air pollution situation in the Philippines, focusing on Metropolitan Manila. Then, it reviews the epidemiological studies on ambient air pollution, justifies the use of the environmental health risk assessment approach based on epidemiological studies, and discusses the risk assessment approach in further detail. Heterogeneity of response to air pollution or a variation in 'vulnerability' is then addressed. Aside from age, the definition of the vulnerable groups will be explored in terms of socio-economic status as a marker of population heterogeneity. This discussion on vulnerable groups highlights the importance of considering the aforementioned variable in health risk assessment. Conclusions are then drawn from this review and aims/objectives are formulated followed by the methodology.

Chapter 1

BACKGROUND AND RATIONALE

1.1. Air Pollution in the Southeast Asian and Western Pacific Regions

Approximately 1.3 billion people live in urban settlements in developing countries and up to 60% of them may be living in conditions that put their health at risk (*WB, 1996*). However, this does not mean that the other 40% do not share some health risk as well. In another estimate, by the year 2000, it is projected that 50% of the world's populations will be living in urban centres and that approximately 50% of these city dwellers will be in the slum and shanty-towns, which in some countries maybe as high as 79% (*Donahue, 1982; UNCHS and WB, 1995*).

Although Asia remains largely rural and agriculture-based, by 1990 32% of its population were estimated to be living in urban areas that, in absolute terms, amounts to almost a billion people (*UNCHS, 1996*). Projections to the year 2000 in Southeast Asia put the urban population at 35% of the region's total population. In the Philippines, its urban population has grown from 32% (of the total population) in 1970 to 49% in 1990. Of this total Philippine population, 15% live in the three biggest cities that include Metropolitan Manila (*WB, 1996*).

With increased urbanization (*Ibid.*) and advances in industrial development in the city environment, several associated hazards in the outdoor environment are apparent. The most serious of these hazards are water pollution, collection and management of solid wastes, noise pollution and air pollution. These hazards are quite common in cities of developing countries as well. The epidemiologic transition, which is the shift from infectious diseases as the most common causes of death, to the non- communicable types of diseases, has started to occur only in the past 30 to 40 years in developing countries. However, unlike in the developed countries, infectious diseases have persisted together with the emergence of the non-infectious, though not for entire populations. Hence, certain subgroups of populations in the developing countries are caught within

this overlap. Underlying this occurrence are changes in the risk factors that go with development and industrialization (*Smith, 1997*). Such subgroups are exposed to both risks of infectious diseases and malnutrition, lifestyle and environmentally related illnesses. In the cities of developing countries, like Metropolitan Manila, these subgroups are mostly the urban poor (*Harpham, 1986*).

Cities are often associated with air pollution, due both to industries and transport vehicles while in the rural areas, the main sources are industrial and domestic in nature. Domestic sources include cooking, both indoors and outdoors, and burning of rubbish and other solid wastes. Due to the economic boom of the early 1990's in Southeast Asia, the problem of air pollution has become especially worse. In certain cities, particularly Bangkok, Jakarta and Manila, the most common and serious air pollutants of concern are suspended particulates, and airborne lead. These two pollutants were ranked first in comparative risk assessments conducted in these cities.(*Ostro, 1994;USAID, 1990; WB, 1996*) In fact in all these three cities, suspended particulates exceeded the WHO guidelines by more than a factor of 2. In addition, carbon monoxide and ozone levels in Jakarta are measured to be above the WHO air quality guidelines (*UNCHS, 1996*). In Bangkok, risks from different environmental problems were ranked. Out of the three major higher risk environmental problems of the city, two pertain particularly to air pollution. It was estimated that up to 1400 deaths maybe attributable to particulate matter alone (*USAID, 1990*). This type of assessment would even be more interesting if it is known which subgroups of the population get the bulk of the health impact of such an environmental pollution.

Even with the downturn of the economies in this region in the past two years, ambient air pollution and its subsequent effects on health remains a major problem for the mega-cities of Southeast Asia that must be addressed accordingly.

1.2. Air Pollution, Health and the Urban Environment of Metropolitan Manila

Metropolitan Manila, or Metro Manila, is located on the south-western coast of the island of Luzon around the mouth of Pasig River which drains into the Manila bay. The total land area is about 636 square kilometres and about 37% of this land area are used for housing which includes single and multiple

residential units and slum and squatter areas. In the year 2000, approximately 12.7% of the population of Metro Manila were estimated to be at or below the poverty line that was equivalent to $450 per annum per person. This percentage is equivalent to more than 211,000 families and has increased from about 8% in 1995. It is likewise estimated that 35% of the population live in slum settlements or squatter areas. The "income gap ratio" in Metro Manila is about 22.1%. This means that the income of those below the poverty line would have to be raised on average by 22.1 percent to reach the poverty threshold. This ratio has increased from 1995 which was about 18%.

Poor housing areas with high density populations are usually located around industrial, commercial and tourist areas that provide formal and informal employment for the residents. The rest of the land area is used either for commercial or industrial or government services, i.e. buildings and offices, streets etc. The more affluent residential areas are located in the southern portions of Quezon City, at Greenhills in San Juan, in the western area of Mandaluyong and Pasig, and around the business district of Makati. All of these affluent areas are situated around Epifanio Delos Santos Avenue (EDSA) which is the principal circumferential 12-lane highway running in a semicircle with a radius of seven kilometres. In the past decade, more affluent neighbourhoods have been established in the south of Metro Manila, particularly in the cities of Muntinlupa and Las Piñas.

Industrial establishments have occupied approximately 15% of the land area of Metro Manila. These factories are located in areas with good transportation links like the port area, the districts of Paco and Pandacan in the city of Manila, and in the some areas of Pasig and Mandaluyong. The number of businesses and industries had increased considerably in the past decade. In 1997, there were about 26,500 industries in the whole Metro Manila. These were concentrated in the north of the metropolis especially in the north of Quezon City and in the cities of Caloocan and Valenzuela. (*Shah et. al., 1997; NSO, 2000*)

With regards to transportation, there was a rapid increase of road vehicles from less than a million in the late 1980's to 2.3 million in 1994. In Metropolitan Manila, the number of vehicles increased from about 600,000 to a little less than

a million from 1990 to 1994, and has continued to grow in more recent years. The increase was more noticeable among the diesel-fuelled vehicles. The number of diesel fuelled vehicles increased from 172,000 to more 301,000 in that time period. Fuel consumption also increased: for gasoline, the increase was from less than 500,000 litres in 1990 to almost 700,000 litres in 1994 and for diesel, it was from 180,000 litres to more than 300,000 litres. In the busiest road networks, traffic peaks at 11,000-12,000 vehicles per hour and daily volumes could exceed 140,000 –150,000 vehicles. In many sections of EDSA, for example, 2.34 million passengers travel daily, of which 1.43 million travel by buses which are mostly diesel fuelled.

Road vehicles contribute substantially to particulate matter loads, though no estimates of emissions have been made recently. Changes in air quality had not been adequately and reliably documented before 1986. Since 1986, TSP pollution had shown no overall trend but the annual average has consistently remained up to five times higher than the WHO air quality guideline. In the past five years, the ambient monitoring data in Metropolitan Manila has consistently measured PM_{10} levels to be above the air quality guidelines. Personal monitoring measurements done in conjunction with an epidemiological study of jeepney and bus drivers and commuters in 1991 and schoolchildren in 1993 showed that levels of total suspended particles, sulphur dioxide, carbon monoxide, total oxidants and lead were way above the WHO air quality guidelines (*WB, 1996*).

In a recent review of the state of environmental health in the Philippines that reviewed available studies and data, four main problems were identified. Two of these main problems were related to ambient air pollution namely, dust-related diseases such as bronchitis, and lead poisoning particularly in Metropolitan Manila. Indoor air pollution was likewise considered. However, with a recent study on indoor exposure to particulate matter, 10μm (PM_{10}) and nitrogen oxides (NO_x) in Metropolitan Manila showing no significant difference in the pollutant levels between inside and outside the homes, this particular aspect was deemed to be a lesser problem (*Soutar, 1999*). Another problem cited in the review was the persistence of diarrhoea and skin diseases due to inadequacy of water supply and sanitation based on an epidemiological study which examined the association between the services available and the diseases. Also cited was

pesticide-use related morbidity based on acute poisoning data from the Poison Control Centre and extent of usage of pesticides (*WB, 1996*). This inadequacy of water supply and sanitation is invariably distributed in the metropolis. Areas such as the Tondo district in the city of Manila, with a large squatter settlement, had incidence of diarrhoea for both adults and children at least two times higher than in the wealthier sections of Manila (*WRI, 1996-97*). As seen from this review, combinations of communicable and non-communicable diseases are attributed in part to environmental pollution. The review concluded that ambient air pollution especially in the Metropolitan Manila area is one of the most important environmental health problems in the Philippines. This important environmental health problem is the topic of this project.

A few epidemiological studies have been done with regard to air pollution. These studies have also shown that the probable morbidity effects of air pollution were unequally distributed among the population who were studied. In a study of jeepney drivers, air-conditioned bus drivers and office commuters, for example, after controlling for several variables such as smoking and educational attainment, it showed that the jeepney drivers had more than twice the odds to have chronic obstructive pulmonary disease as compared to the commuters who were mostly office workers. Jeepneys are passenger vehicles that could sit about 10 to 14 passengers at one time. Jeepney drivers were more exposed to air pollution due to the nature of their job and were generally less educated than the commuters. (*Subida et. al., 1991*)

Three groups of children were likewise surveyed separately with regards to respiratory health and air pollution in the early 1990's. These were the child scavengers, child vendors and schoolchildren. Of the three groups, the child scavengers were shown to have the most respiratory symptoms and higher prevalence of abnormal pulmonary function test results. They were also generally malnourished compared to the schoolchildren and child vendors. These child scavengers had the highest blood lead levels, most of whom exceeded the WHO guideline followed by the child vendors and then the schoolchildren. Child scavengers lived near the dumpsite where they scavenge in squatter communities. Exposure levels of child scavengers to total suspended particulates, carbon monoxide and lead were two to three times higher than the

schoolchildren. Most child vendors live also in poor areas but most of them study as well before going out in the streets to sell flowers, candies or cigarettes for three to four hours a day. Compared to the schoolchildren, the child vendors had twice as much respiratory symptoms and abnormal pulmonary function test results. The schoolchildren, aged 6-10 years old, had the lowest prevalence of respiratory symptoms and low pulmonary function test results among the three groups of children. The schoolchildren are the least exposed to ambient air pollution (*Subida et. al., 1994; Subida et. al., 1991*). From these studies especially on the jeepney drivers, it is suggested that exposure to air pollution could affect respiratory health negatively. Furthermore, it is also suggested that an unequal exposure to environmental pollution, in this case air pollution, of sub-populations based on the socio-economic situations exists in the metropolis.

1.3. The Epidemiological Evidence

This section presents evidence from epidemiological studies about the effects of air pollution especially particulate matter on morbidity and mortality. The Medline and PUBMED databases were used extensively to search for epidemiological studies relevant to this study. In addition, relevant internet websites were also searched. The search included studies pertaining largely to particulate matter and mortality and to a lesser extent, morbidity. Approximately more than 150 papers were reviewed for this purpose. Only those papers in the past 10 years were considered, however, the exact number of papers can not be ascertained at this point in time anymore. The focus of this project, as will be evident later, is the effects of air pollution on mortality although in this review morbidity studies are likewise cited. The morbidity studies are discussed only to illustrate further the extent of the effects of air pollution in humans in different parts of the world. The following presents evidence for effects of particulate matter followed by a detailed review and critique of these mortality studies.

1.3.1. Ambient Air Pollution: Mortality Studies

A large number of epidemiological studies have come out in the past 20 years regarding the effects on morbidity and mortality of environmental pollution in general and air pollution in particular. Of particular significance are three studies carried out in the United States permitting the estimation of lifetime risks of total and cardio-respiratory mortality from particulates and other pollutants.

Factors such as the effects of smoking, socio-economic status and occupational exposures were often controlled for in these studies. Many others had also looked at the effects of pollutants on daily mortality in different cities.

Effects of air pollution on daily mortality, particularly respiratory and cardiovascular, have been studied quite extensively. Schwartz argued that the pattern of results of such studies, based on his review and meta-analysis, could reasonably be interpreted as a causal association. A relative risk of 1.06 per 100 $\mu g/m^3$ of total suspended particulates was found (*Schwartz, 1994*). Dockery and Pope, in their own review and combining several studies, have found a 1% change in daily total mortality for each 10 $\mu g/m^3$ increase in PM_{10}, a 3.4% change in respiratory mortality and a 1.4% change in cardiovascular mortality (*Dockery et. al., 1994*). Ostro, in combining five studies, found a relative risk of 1.23 change in daily mortality for each 10 $\mu g/m^3$ change in PM_{10} (*Ostro, 1996*). A more recent review of studies from 20 and then the 90 largest cities of the United States by the Health Effects Institute found an average estimate of about 0.5% increase in all cause mortality for a 10 $\mu g/m^3$ increase in PM_{10} (*Samet et. al., 2000, Dominici et. al., 2000*). Dab et al recently conducted a critical appraisal of the major review articles of time series studies looking at the relationship between particles and mortality, specifically cardiopulmonary mortality. Arguments from 15 reviews were included and assessed in the study to investigate whether the association between air pollution and health was just a correlation or most probably causal. Their methods involved distinguishing validity from causality and systematically analyzing the arguments presented by the 15 review articles according to the causality and validity criteria. The authors concluded from this comprehensive and critical analysis that the observed association in the time series studies reviewed was, indeed, valid and fulfilled the criteria for causality (*Dab et. al., 2000*). In other studies of the acute effects of particles on daily mortality, a range of 0.7 – 1.6% change in daily total mortality for each 10 $\mu g/m^3$ increase in PM_{10} was found. In a pooled estimate of four studies from the United States and one from Chile, a 1.23% change in daily mortality for each 10 $\mu g/m^3$ change in PM_{10} was also found. For respiratory mortality, the values were higher at 1.5-3.7%. For cardiovascular mortality, the range was from 0.8% for haze in the Sta. Clara study (*Fairley, 1990*) to about 1.8% for PM_{10} in the Utah Valley study (*Pope et. al., 1992*).

To show the geographic diversity of the time series studies conducted, the following table gives some examples of recent studies using this study design.

***Table 1.1**: Mortality and Pollution time series studies*

YEAR	AUTHORS	LOCATION	FINDINGS/COMMENTS
1994	Schwartz	Cincinnati, Ohio	Study period:1977-82 Daily Mortality RR for 100 μg.m^{3}increase in TSP was 1.06 (1.03-1.10). RR for elderly was higher (1.09).For Pneumonia (1.18) and CVD (1.08)
1995	Saldiva, Pope, Dockery, Schwartz et al	Sao Paolo, Brazil	Study period: May 1990-April 1991. Increase of 100 μg/m^3 in PM_{10} was associated with 13% increase in daily mortality for the elderly (=/>65 yrs old). Dose-response relationship was almost linear.
1996	Verhoeff, Hoek, Schwartz, van Wijnen	Amsterdam, Holland	Study period: 1986-92 Relative risk of 1.19 was associated with increase of 100 μg/m^3 of black smoke, 1.06 for PM_{10}. RR for the elderly (>64 yrs old) was higher at 1.26 for black smoke and 1.07 for PM_{10}.
1996	Ballester, Corella, et al	Valencia, Spain	Study period: 1991-93 The RR of dying due to 10 μg/m^3 increase in mean daily black smoke was 1.009 and for aged 70 years and over, it was 1.008. Cardiovascular mortality was at 1.012
1997	Kelsall, Samet, Xu and Zeger	Philadelphia	Study period: 1974-88 1% increase in mortality was associated with the interquartile range increases of 34.5 μg/m^3 of TSP, and 12.9 ppb of SO_2 . For ozone, it was 2% increase in mortality for 20.2 ppb.
1998	Ponka, Savela, et al	Helsinki, Finland	Study period: 1987-93 3.5% increase in total mortality and 4.1% increase in cardiovascular mortality were associated with an increase of 10 μg/m^3 of PM_{10} for persons aged 65 years or less. Ozone had an independent effect as well.
1998	Zmirou, Schwartz, et al	10 European cities	Meta-analysis results: For 50 μg/m^3 increases of black smoke, RR of cardiovascular conditions was 1.02 and for respiratory diseases, 1.04, for the western European cities. For SO_2, the RRs were 1.04 and 1.05 respectively.
2000	Hoek, Brunekreef et al	4 major urban centres in the Netherlands	Study Period: 1986-1994 Relative risk of 1.02 for total mortality for increase of 100 μg/m^3 PM_{10}. Pneumonia deaths had the highest relative risks Higher relative risks of total mortality for PM_{10} seen in the summer than the winter. Other pollutants had consistent findings as that of PM_{10}
2001	Lee, Kim, Hong et al	7 major cities of Korea	Study Period: 1991-1997 Increase of 0.5-4% in all cause mortality with increase of 100 μg/m^3 TSP. For SO_2, increase of 1-12% mortality corresponds to increase of 50 ppb SO_2.

Comparative table showing the geographic diversity and results of some time series studies conducted on Mortality and Air Pollution. *(Scwartz, 1994; Saldiva et. al., 1995; Verhoeff et. al., 1996; Ballester, et. al. 1996; Kelsall, et. al. 1997; Ponka et. al. 1998; Zmirou, et. al., 1998; Hoek, et. al., 1998; Lim, et. al., 2000)*

Population-based ecological studies of mortality rates by state or area versus pollution indices of chronic exposure to particulate pollution have been reviewed by Dockery et al. (*Dockery et. al., 1996*). The studies have shown consistent results regarding the positive association between mortality and air pollution. However, as pointed out in the review, due to the studies' 'ecologic' design which is vulnerable to unmeasured confounding, and the size of the estimated association which is quite unconvincing, these studies may not be considered definitive.

More reliable for estimating the mortality effects of chronic exposure to particulate pollution are the two prospective cohort studies done in the US by Pope et al, and Dockery et al. In the six US cities study, a 14-16 year mortality follow-up was conducted among 8,111 adults. Individual risk factors such as smoking status, sex, age and others were controlled for. Statistically significant associations were found between air pollution and mortality. Comparison of the most polluted to the least polluted cities revealed a relative risk of 1.26. Positive associations were also found between cause-specific deaths such as lung cancer (RR-1.37) and cardiopulmonary diseases (RR-1.37), and fine particles pollution (*Dockery et. al., 1993*). An American Cancer Society study conducted by Pope et al involved 151 US metropolitan cities where some 552,138 adult-subjects lived. The subjects were followed-up from 1982 to December 1989. Smoking, education and other risk factors were included in the analysis. Although the result was small compared to deaths associated with cigarette smoking, a positive association was found between mortality and particulate air pollution by the six-cities studies. Relative risks for total mortality, cardiopulmonary disease and lung cancer were 1.17, 1.31, and 1.03 respectively (*Abbey et. al., 1999*). Although, the exposure data used by these two studies are still 'ecologic' in nature, the control of individual risk factors especially that of smoking, made the estimates of associations more dependable compared to the population-based cross-sectional studies.

Abbey et al reported a third cohort study on mortality and air pollution. A cohort of 6,338 non-smoking residents of California who were members of the Seventh Day Adventists Church was included in the study. This cohort was followed from 1977 to 1992 and their mortality patterns were assessed in terms of

PM_{10} and other air pollutants. Several covariates were controlled namely age at baseline, pack years of past smoking, years lived/worked with a smoker, education, occupation and occupational exposures, and body mass index. The results found associations of PM_{10} above 100 $\mu g/m^3$ with both non-malignant respiratory disease and lung cancer. Ozone and sulphur dioxide also showed similar results. For males, significant associations were seen between all natural cause mortality and PM_{10} above 100 $\mu g/m^3$, which remained stable even with the addition of the other pollutants in the model, relative risk of 1.12 (1.01-1.24). For females, no significant associations were seen. These significant associations between PM_{10} and mortality were based on the days when PM_{10} levels were above 100 $\mu g/m^3$. For mean concentration difference in interquartile range, no significant differences were found. (*Ibid.*) Some of these findings were quite consistent with the other two cohort studies. Its main advantage, however, is that smoking could not be considered a confounder. Nevertheless, the transferability of its results may be more problematic as the cohort's lifestyle characteristics is less representative compared to the other cohort studies. (*Hurley et. al., 2000*)

Studies linking air pollution with child mortality are very few and are limited to infant mortality. Two notable ecologic studies have been done in Rio de Janeiro, Brazil and in the Czech Republic in 1991 and 1992 respectively. The Brazil study, using aggregate data at the city level, showed an association between air pollution and infant pneumonia mortality but not for the general infant mortality nor for infant mortality due to diarrhoea. Similar results were found by Bobak and Leon in the Czech Republic where post neonatal respiratory mortality risk ratios between the highest and lowest quintiles were found to be 2.41 for TSP, 3.91 for SO_2 and 1.20 for NO_x (*Penna et. al., 1991; Bobak et. al., 1992*). These studies present evidence regarding the susceptibility and vulnerability of this particular age group.

Two significant studies on infant mortality and air pollution have been published recently. Woodruff et al evaluated the relationship between post neonatal mortality and particulate pollution in the United States among 4 million infants born from 1989 and 1991, from 86 metropolitan areas. Exposure data was ascertained from the USEPA and exposure categories were divided into high, medium and low levels of PM_{10} pollution. Their results, after controlling for other

demographic and environmental covariates, showed overall post neonatal mortality odds ratios of 1.05 and 1.10 for babies in the medium and high pollution categories respectively, as compared to the low pollution category. Further analysis into respiratory deaths alone showed higher odds ratios for both normal and low birth weight babies. The odds ratio for a continuous 10 $\mu g/m^3$ change in PM_{10} was 1.04 (*Woodruff et. al., 1997*). In the Bobak case-control study, 2,494 infant deaths with data on exposure were investigated. The results of the study suggested that air pollution effects on infant deaths maybe specific to respiratory causes. Rate ratios drawn from the study was only significant for respiratory post neonatal mortality at 1.95 per 50 $\mu g/m^3$ increase TSP and 1.74 for SO_2, adjusted for socio-economic factors and birth weight (*Bobak et. al., 1999*). The above findings imply positive and independent association between infant deaths and air pollution apart from the effects for the total population.

***Table 1.2**: Comparative overview of Cohort Mortality Studies Designs*

ADULTS							
Study and setting	**Sample size**	**Sample design**	**Sampling Frame**	**Outcome Measures**	**Exposure measures**	**Response rate and refusers**	**Other risk factors accounted for**
Dockery et al (1993) 6 US cities	8111	Random sampling for 6 communities. Limited to white 25 to 74 yrs old	Census tracts	Basic characteristics were ascertained by informational letters and postcards mailed every year . Death certificates, coding was done blindly	Centrally-located monitoring station for each community measuring fine and inhalable particles.	98% of death certificates were obtained and coded. For all those lost to follow-up, person-years of contribution to the study were calculated and included in the analysis. For those who did not respond, family members , friends and neighbours were questioned	Age, sex, active smoking, education, body mass
Pope et al (1995) 151 US metropolitan areas	552,138	Recruited by ACS volunteers drawn from an ongoing cohort study. Included are those at least 30 years old	ACS Cancer Prevention Study w/c involved 1.2 M individuals	Basic characteristics were obtained by questionnaire. Death certificates, coding was done blindly	Estimates were used from the USEPA national aerometric data base and the from the EPA dichotomous sampler network.	Death certificates were obtained in 96% of deaths. 2% were lost to follow-up but were traced through the National death index.	Age, sex, race, active and passive smoking, education, body mass, alcohol intake and occupational exposure

***Table 1.2**: Comparative overview of Cohort Mortality Studies Designs (Continued)*

INFANTS							
Study and setting	**Sample size**	**Sample design**	**Sampling Frame**	**Outcome Measures**	**Exposure measures**	**Response rate and refusers**	**Other risk factors accounted for**
Woodruff et al (1997) 86 US metropolitan areas	4 million infants	Listed from the National Centre for Health Statistics .	NCHS - All infants in the 86 areas were included	Information on Post neonatal Infant outcome and maternal and infant characteristics were obtained from NCHS-linked birth/infant death data files for 1989-1991.	Air quality data from the EPA. An infant's exposure was considered to be the mean of the PM_{10} levels for the first 2 months of life	States which had no data on maternal education and maternal smoking were excluded from the study. Also, files with geographic information were not included. These were infants residing in communities with <100,000 population.	Adjusted for maternal education, maternal race, parental marital status, birth weight and maternal smoking during pregnancy, month of birth, ambient temperatures
Bobak et al (1999) Czech Republic	2,594 cases and 35, 642 controls	Qualified post neonatal deaths were included as cases. 14 controls for every 1 case.	From the birth and death registratio n systems	Information on infant outcome and maternal and infant characteristics were provided by the birth and death registry	Air quality from the 46 monitored districts. TSP, SO_2 and NO_x were considered but only TSP was found to be correlated	86% of all births were successfully linked to the death registry. The number of cases was also limited by the availability of pollution data.	Adjusted for maternal age, education, marital status, parity, birth weight, length and gestational age.

(Pope et. al., 1995; American Thoracic Society, 1996, Dockery et. al., 1993, Bobak et. al , 1999)

The Pope study has clear advantages relative to the Dockery study. The Pope study had a bigger sample size and had controlled for more individual characteristics. However, both had limitations with regard to exposure measurement since both relied upon air quality measured at a single station in each metropolitan area during the period of follow-up and also, neither study considered earlier exposures. Using the city level exposure data instead of individual exposure levels would lead to some misclassification error, a common occurrence in air pollution studies.

The Pope study is extensive with more than 500,000 people included and 151 metropolitan areas from 48 states of the United States. Also, aside from its bigger sample size, the Pope study also controlled for more potential confounding factors. On the other hand, the Dockery study is quite limited with only six cities which are mostly located in the north-eastern part of the country. For the Dockery study a relative risk of 1.26 (CI 1.08,1.47) was seen for a difference of 18.6 $\mu g/m^3$ $PM_{2.5}$ while 1.17 (CI 1.09,1.26) was seen for the Pope study for a difference of 24.5 $\mu g/m^3$ $PM_{2.5}$. (*Pope et. al., 1995; Dockery et. al., 1993*) Although, it

is apparent that the Pope study is more extensive, an analysis of the two studies producing a pooled estimate was performed in this study, description of which is found in Chapter 2. The use of a pooled estimate from several epidemiological studies is recommended by the WHO guideline document on Environmental Health Risk Assessment. (*WHO, 2000*) The pooled estimate, as expected, resulted in a relative risk much nearer the ACS-Pope study relative risk, 1.05 (CI 1.03, 1.07) increase in mortality per 10 $\mu g/m^3$ PM_{10} increase.

A reanalysis of these two cohort studies was recently undertaken. There were two parts to the reanalysis. The first part involved the replication and validation of the original studies and in the second part, the assessment of the robustness of the original analyses by using alternative risk models and analytic approaches. For the second part, several variables such as passive smoking, marital status and alcohol consumption to name a few, which were available but were not used in the original studies were included in the model. However, in spite of these variables, the results were still similar to the original. In addition, the reanalysis also included testing of the effects on susceptible subgroups of the population. A relative risk of 1.28 for a difference of 18.6 $\mu g/m^3$ $PM_{2.5}$ for the Dockery study and 1.18 for the Pope study for a difference of 24.5 $\mu g/m^3$ $PM_{2.5}$. Both of these estimates were very close to the estimates in the original publications. A most interesting aspect of the reanalysis is the investigation into the effects of the particulates and sulphates among different potentially susceptible subgroups in the population. The subjects were classified according to age, gender, smoking status, educational level, and occupational exposure to dust or fumes and marital status. Variation in the estimates were seen in most of the different subgroups, however, the estimates were not statistically significant except for the educational level which is an indication of socio-economic status. The results showed that the relative risk of mortality associated with an increase in fine particulates was higher for those who were less educated. The relative risks were 1.35 (1.17,1.56), 1.23 (1.07,1.4) and 1.06 (0.95,1.17) for subjects with less than high school education, high school and more than high school education respectively. These are for a difference of 24.5 $\mu g/m^3$ PM_{10}. (*Krewski et. al., 2000*) This finding could be very important as regards policy or programme formulation as it identifies a specific subgroup of the population which could be more susceptible or vulnerable to air pollution. These socio-economic differences

were considered in estimating mortality due to particulate pollution in this assessment.

For the infants, the estimates from Woodruff et al and Bobak were similar. Transforming the Bobak estimate of 1.12. per 50 ug/m^3 TSP to per 10 ug/m^3 PM_{10} would yield a risk of 1.042 per 10 ug/m^3 PM_{10} increase. These estimates of excess risk in the infant mortality study are almost similar with that seen in the adult cohort studies even if the time of exposure was shorter. According to Pope, this indicates that the relevant time of exposure is much shorter and/or that infants are at higher risk for exposure to air pollution. (*Pope, 2000*) The relative risk for infants from the Woodruff study was 1.04 (CI 1.02,1.07) for an increase of 10 µg/m^3 PM_{10}. (*Woodruff et. al., 1997*)

ADULT COHORT STUDIES:

a. Six-Cities study by Dockery et al:

Relative Risk for a difference of 18.6 µg/m^3 $PM_{2.5}$ is 1.26

18.6 µg/m^3 $PM_{2.5}$ = 26.57 µg/m^3 PM_{10}

((Log 1.26)/26.57) x10 = 0.0.08698, exponentiated to 1.09

1.09 (1.03-1.16) per 10 µg/m^3 PM_{10} increase

b. American Cancer Society Study by Pope et al:

Relative Risk for a difference of 24.5 µg/m^3 $PM_{2.5}$ is 1.17

24.5 µg/m^3 $PM_{2.5}$ = 35 µg/m^3 PM_{10}

((log 1.17)/35) x10 = 0.044857 exponentiated to 1.046

1.046 (1.025-1.068) per 10 µg/m^3 PM_{10} increase

*The confidence intervals were calculated similarly.

In time series studies, the timing of death is studied according to air pollution events and temporal trends within a defined geographical area. However, most of these studies are not capable of determining the degree of premature death beyond a few days ('harvesting effects') (*McMicheal et. al., 1998*) or a few weeks (*Schwartz et. al., 2000*). Therefore, for the purpose of this risk estimation where life expectancies and person years of life lost are calculated, evidence derived from the time series studies will not be used directly to estimate the mortality rate ratios but to quantify a risk modifier such as age and assess whether geographic location or exposure differences affects exposure-response

relationships. The arguments regarding the choice of the type of studies to be used in risk assessment are presented in a later section in this chapter.

Systematic reviews of time series studies relating daily mortality and morbidity with air pollution levels have been done with both US and European studies. In his meta analysis of 13 studies made mostly in the USA, Schwartz outlined the principal concerns when reviewing time series studies. These concerns are the following:

1. Failure to adequately control for long term fluctuations in daily mortality and daily air pollution. This refers primarily to seasonal influences and other such factors.
2. Confounding due to weather factors
3. Potentially nonlinear dependence of daily mortality on air pollution
4. Possibility that the observed relationship could be driven by a few extreme days.

Furthermore, due to slight differences in the quality of the studies reviewed, he proposed to do a quality-based meta analysis, although an equal quality weights meta analysis was also made. The basis of his quality ranking scores were the consideration of other pollutants, potential non-linearities in the weather dependence of mortality and the measure of exposure (TSP vs PM_{10} vs $PM_{2.5}$) (*Schwartz, 1994*).

Likewise, Thurston and Kinney, proposed guidelines or criteria by which these type of studies must be made and/or evaluated that were similar but more detailed than those of Schwartz based on factors which affect results of time series studies. The factors referred to are:

1. long wave influences on the data
2. model specification
3. data distribution effects i.e. over dispersion

The general guidelines they proposed are the following:

1. Inclusion of all potential confounders in the analysis except those which are not biologically plausible.

2. Pollutant measurements should be representative of the exposure of the population and when available, a non-respiratory or non-cardiovascular outcome must be included as control
3. Exploratory statistical analysis should be conducted prior to statistical modelling, potentially including intercorrelations of the x term, variable cross-correlations and autocorrelation functions for all y and x terms. Also autocorrelation in model residuals should be evaluated via the Durbin Watson (DW) statistic.
4. If present, autocorrelation should be addressed by appropriate methods possibly including an analysis of the data by season, data prefiltering, the inclusion of model terms for all possible long wave cycles, and/or autoregressive methods.
5. Intercorrelations among the coefficients of model variables should also be evaluated to aid in the interpretation of model results.
6. Ordinary least square model residuals should be checked for deviations from normality (W) and if present, nonnormalities should be addressed (via Poisson modelling for count data).
7. It is desirable for such analysis to include a sensitivity analysis inter-comparing various models and model specifications. (*Thurston et. al., 1995*)

It is with these guidelines, that a systematic review of studies from developing countries is made in the next section.

A most comprehensive review and pooled analysis of time series studies was recently done in the United States of 90 cities (and within those the 20 largest) initiated by the Health effects Institute. Selection of the cities was based on population size and availability of PM_{10} data. There were two stages of analyses done. The first stage involved the individual city modelling. The model developed was a log-linear generalized additive model accounting for the smooth longer-term fluctuations in mortality. The smoothing functions were introduced to control for potential confounding by longer-term trends and by shorter-term factors as well. In the second stage, hierarchical regression models combining the associations in the individual cities were used. The results of these multi-city studies revealed an average estimate of 0.5% increase in the overall mortality for

a 10μg/m^3 PM_{10} increase with a 1day lag. The 20 largest cities specifically showed a 0.48% (95% interval, 0.05, 0.92) increase in mortality with the 10 μg/m^3 PM_{10} increase on concurrent day. The adjustment for Ozone and any other pollutant had little effect on the magnitude of this estimate. This suggested that PM_{10} effect is robust to the inclusion of other pollutants. In addition, the relative risk coefficients for the respiratory and cardiovascular deaths were found to be slightly greater than the total death (*Samet et. al., 2000; Dominici et. al., 2000*). However, although analysis of elderly hospitalization was done in 14 of the cities, separate analysis for elderly mortality was not.

For the present assessment, a pooled estimate from a meta-analysis of three studies from Holland, Spain and Cincinnati, USA for the total and elderly relative risk are computed (*Schwartz et. al., 1994; Verhoeff et. al., 1996; Ballester et. al., 1996*) The relative risk for all ages is 1.009 (1.005-1.012) and the elderly relative risk is 1.011 (1.005-1.016) per 10 μg/m^3 PM_{10}. As mentioned earlier, both estimates are used for approximation of the different relative risks between younger and older age groups. This will be explained later.

The following is a review of seven different time series studies conducted in cities of developing countries namely Santiago, Chile, Mexico City, Mexico, Beijing, China, Sao Paolo, Brazil, New Delhi, India and Bangkok, Thailand (*Xu, et. al., 1994; Gouveia, 1997; Borja-Aburto, et. al., 1998; Cropper et. al., 1997; Chestnut et. al., 1998; Ostro et. al., 1995; Loomis et. al., 1999*). There has been no systematic review of such studies published. The table outlines the most important aspects of time series studies that must be considered based on the recommendations of Schwartz, Thurston and Kinney. Discussions further on describe other aspects of the individual studies as suggested by the guidelines.

Table 1.3: Time Series Studies in Developing Countries

Study, Setting and Period	PM monitoring	Other Pollutants modelled	Long wave/ Autocorrelation addressed	Data distribution (over dispersion) addressed	Lags addressed	Multiple models	Other Limitations	Relative Risks/percent change obtained
Ostro et al, Santiago, Chile, 1989-91	5 sites, PM_{10}	O_3, SO_2, NO_2	yes	yes	Poll = 0-3d Temp=/< 3d	Yes	Complete April-November exposure data , rest of the year – lots of missing data, humidity not included, trauma causes of mortality included	1.1% /10µg/m^3 PM_{10} for total mortality
Gouveia, Sao Paolo, Brazil, 1991-93	4 sites, PM_{10}	SO_2,NO_x, CO, O_3	yes	yes	Poll = 0-2d Temp=/< 5d	yes	Only elderly mortality and under 5 mortality, no total mortality, trauma causes of mortality included	0.5%/10µg/m^3 for elderly mortality (>65 yrs)
Borja-Aburto et al, Southwest Mexico City, Mexico, 1993-95	1 site, $PM_{2.5}$	NO_2, O_3	yes	yes	Poll = 0-4d Temp=/< 6d	yes	Single site monitoring	1.68% /10ug/m^3 $PM_{2.5}$ for total mortality. 2.27% for elderly mortality (>65yrs) 1 68% for respiratory causes and 3.42% for cardiovascular causes
Loomis et al, Mexico City, Mexico, 1993-95	1 site, $PM_{2.5}$	NO_2, O_3	yes	yes	Poll = 0-5d Temp=/< 6d	Yes	Single site monitoring	6 87% excess infant mortality/10 ug/m^3
Xu et al, 2 residential areas in Beijing, China,1989	2 sites, TSP	SO_2	yes	yes	Poll=0-5d Temp = not stated	No	Incomplete data set for pollution levels	4% total mortality/ 100ug/m^3 TSP – but not significant.(effect seen only on summer months)
Cropper et al, New Delhi, India, 1991-94	9 sites, TSP	SO_2	yes	yes	Poll = 0-3d Temp=/< 7d	Yes	Only 25% of total mortality of the area was considered .	All figures are per 100ug/m^3 change: 2.3% Total Deaths 4.3% CVD deaths 3.1% Resp death By Age grp: 0-4: 2.4% 5-14: 2.6% 15-44: 4.3% 45-64: 2.0% >65:).8%

Table 1.3: Time Series Studies in Developing Countries (Continued)

Study, Setting and Period	PM monitoring	Other Pollutants modelled	Long wave/ Autocorrelation addressed	Data distribution (over dispersion) addressed	Lags addressed	Multiple models	Other Limitations	Relative Risks/percent change obtained
Chestnut et al, Bangkok, Thailand, 1992-1995	4 sites, PM_{10}	none	yes	yes	Poll = 0-3d Temp= 3dMA	No	Limited number of days for which PM_{10} data is available. Less than accurate level of mortality for the years 1994-95.	For estimated PM_{10} data, 1992-93: 1.7% Natural mortality/10ug/m^3 1.2% Cardiovascular mortality 3.8% Respiratory mortality 3.8% for older adult mortality =/>50 yrs old

(Xu et. al., 1994; Gouveia, 1997; Borja-Aburto et. al., 1998; Cropper et. al., 1997;Chestnut et. al., 1998; Ostro et. al., 1995; Loomis et. al., 1999)

Out of the six cities included in this review, only Mexico City monitored $PM_{2.5}$. Although smaller sized particulate matter is said to be more evenly distributed than TSP, having a single site may still be problematic with regards to whether this is representative of the exposure of the population under study considering the vastness of the area and the population distribution. In other cities, multiple sites that were averaged and checked for inter-station correlation were used. Consistency of data from previous years was also examined in the case of Santiago, Chile. In Bangkok, only one of the four sites was used due to missing data from so many days in the other three. However, to ensure reliability of that one station, on the days where data were available for the other three sites, correlation coefficients were computed. Apart from the number of stations, another issue here is the distribution or location of the stations in the geographic areas of concern as this would again have a bearing on the representativeness with regards exposure of the population. The New Delhi study did not mention anything about this. The four stations of Bangkok and Santiago were all located in the centre of the cities that may not necessarily be where most representative of the population under study resides. Only the Beijing study had a description of where most of the study population lives, in this case the majority resides around the two monitoring stations. Thus, as in the studies done in developed countries, generalization of exposure from a single or a few monitoring stations which may or may not be evenly distributed amongst the study population, is problematic. Nevertheless, the results from these studies are fairly consistent with each other.

Except for the Bangkok and Beijing studies, other pollutants were analyzed together with particulate matter (PM) in the other five studies. Attempts were made by the studies to characterize the effect of particulate matter in the presence of other pollutants. As seen in their analysis with two or three pollutant models, the relative risk values or percent change become different as compared to the one pollutant model (except for the Santiago study). In making use of co-pollutants in the models and together with consideration for other factors such as temperature and humidity, confounding is minimized as these other pollutants and factors may affect the outcome. The Bangkok study, which did not consider any other pollutants in their model, is uncertain in this aspect. The Bangkok study is the only time series study that has been conducted in a tropical climate so far. All the studies considered factors that may confound the association such as temperature, humidity and weather patterns of the cities.

An important aspect of time series studies is the control for the effect of seasonal patterns and other variations in the health outcomes which have periodicities longer than just a few days resulting in autocorrelation. Autocorrelation can produce both short and long wave influences in the data so that the immediate or short term results which, in this case, is mortality due to (or at least correlated with) increases in exposure is usually estimated with spurious precision. Therefore autocorrelation can conceal the association between the pollutant and the health outcome. This problem could be assessed by tests such as the Durbin–Watson Statistic, and if it exists, the data must be subjected to prefiltering or by including dependent variables in the regression that may account for the long wave cycles such as seasonal patterns (*Thurston et. al., 1995*). Fortunately all of the studies in this review were able to resolve the autocorrelation issue. By adjusting for the seasonality and cyclical patterns in the data through the use of dummy variables for all possible long wave cycles, autocorrelation was likewise addressed. (*Schwartz, 1994*)

Another important issue in time series studies is over dispersion which refers to a variation in the data that maybe greater than predicted from the classic Poisson distribution (*Chestnut et. al., 1998*). It is also referred to as extra-Poisson variation (*Rothman, 1999*). This issue was dealt with by inflating the estimated variance by an over dispersion factor as was done in the Sao Paolo Study. A

programme using generalized estimating equations dealing with over dispersion was also used (*Gouveia, 1997*). In other studies like the Bangkok study, the extra-Poisson variability was modelled and incorporated in the standard errors estimates, hence addressing the problem (*Chestnut et. al., 1998*).

Most of the studies in this review revealed some difficulties with the data set. Lack of data for both the exposure and outcome variables was the main limitation. In the case of Bangkok, due to the large number of days without data for three of the monitoring stations, only the data from one station were used. Data from this station were also not very complete but better than the other three. This matter was sorted out by the investigators by estimating levels of PM_{10} imputed based on the correlation between the airport visual range and particulate pollution, and comparing the levels of the one used with whatever data there were for the other three for consistency. For the Santiago study, since there were enough observations in the three years, only those with data were considered for the analysis: a total of 790 observations in three years. In the Beijing study, only 194 days out of the whole year were included since levels were collected only for 2 to 3 weeks each month. For the mortality data, the New Delhi study is limited since only 25% of the total mortality was included in the study. It will be difficult to generalize this for the whole city, although the authors of the study contend that the geographic distribution of the Delhi residents, who died of non-traumatic causes in the region where the data were collected, mirrors the geographic distribution of the population. Most of the other mortality data were more or less complete in the other studies. The Sao Paolo, Mexico City, New Delhi and Bangkok studies give us clues as to the effects of particulate matter in different age groups. Following the guidelines listed earlier, it is desirable to include a sensitivity analysis inter-comparing various models and model specifications. Not all of the studies presented here were able to do sensitivity analysis, and those that did, found the models to be robust enough. These were the Santiago, New Delhi, Sao Paolo and Bangkok studies.

The studies that were reviewed have fulfilled many if not all of the items in the guidelines. For the purpose of this study, pooled estimates for all cause mortality and elderly mortality from three studies that have both total and elderly relative risks were calculated and used. This would also follow the WHO

recommendation which prefer to make use of an estimate from several studies instead of just one study (*WHO, 2000*). The Beijing study's best fit for calculating the log relative risk was based on the log of the exposure levels (*Xu et. al., 1994*). This analysis was different from the other studies, hence the exclusion. The pooled estimates calculated were used for the purpose of approximating the difference between the elderly and younger age group mortality using the estimates from the cohort studies.

The following are the table for the calculations for the different developing countries' studies and the summary table of the developing countries' conversions to theta coefficients with the standard error (SE):

***Table 1.4**: Calculations of the theta Coefficients for each city study*

City, Country	Calculations
1. Bangkok, Thailand	Coefficient for All ages – 0.104 (0.028) (3-day lag) >/= 65yrs - 0.10 (0.04) (3-day lag) Figures were per 100 μg/m^3 PM_{10}. Thus conversion to per 10 μg/m^3 is dividing the figures by 10.
2. Santiago, Chile	Coefficient for All Ages - 0.075 (0.013) (1-day lag) >/= 65 yrs – 0.091 (0.017) (1-day lag) Figures were per 100 μg/m^3 PM_{10}. Thus conversion to per 10 μg/m^3 is dividing the figures by 10.
3. New Delhi, India	Coefficient for All Ages - 0.00023 (0.0001)/unit change in TSP (2-day lag) *PM_{10} = 0.55TSP Theta coefficient/unit change TSP divided by 0.55TSP x 10 μg/m^3 PM_{10} 0.00023/ 0.55 = 0.00042 x 10μg/m^3 = 0.0042 SE for PM_{10} = SE for TSP x 0.55TSP 0.0001 divided by 0.55 x 10 = 0.002
4. Mexico City, Mexico	Relative Risk for all ages : 1.0136 (1.0020 – 1.0252) per 10 μg/m^3 change in $PM_{2.5}$ (current and 4-day lag) a. First convert the $PM_{2.5}$ to PM_{10} *$PM_{2.5}$ = 0.7PM_{10} =10/0.7=14.3 Log 1.0136/14.3 μg/m^3 PM_{10} x 10 μg/m^3 PM_{10} = 0.00945 (theta coefficient) b. log 1.0020/14.3 x 10 = 0.00139 c. log 1.0252/14.3 x 10 = 0.01740 Therefore for per 10 μg/m^3 PM_{10} RR= 1.0095 (1.0014 - 1.0175) d. To get the SE : SE = log 1.0175 – log 1.0095 / 1.96 = 0.004031 Relative Risk for >/= 65 YRS 1.0158 (1.0004 – 1.0312) per 10 μg/m^3 change in $PM_{2.5}$ (4-day lag) a. log 1.0158/14.3 x 10 = 0.01096 (theta coefficient) b. log 1.0004/14.3 x 10 = 0.00028 c. log 1.0312/14.3 x 10 = 0.02147 Therefore for per 10 μg/m^3 PM_{10} RR= 1.0110 (1.0002 – 1.0217) d. To get the SE: SE = log 1.0217 – log 1.0110 / 1.96 = 0.0054

Table 1.4: Calculations of the theta Coefficients for each city study (Continued)

City, Country	Calculations
5. Sao Paolo, Brazil	Relative Risk for >/= 65 yrs – 1.033 (1.006-1.060) for a difference in 10^{th} –90thcentile (1-day lag) PM_{10} change from 10^{th} to 90^{th} centile = 64.2 $\mu g/m^3$ To convert to per 10 ug/m^3 PM_{10} a. Log 1.033/64.2 x 10 = 0.005057 (theta) b. log 1.006/64.2 x 10 = 0.00093 c. log 1.060/64.2 x 10 = 0.00908 Therefore for per 10 $\mu g/m^3$ PM_{10}:, the relative risk is 1.005 (1.0009 – 1.009) d. To get the SE SE = log 1.009 – log 1.005 / 1.96 = 0.00205

*0.55TSP = PM_{10} (PEHAS Study) *0.7PM_{10} = $PM_{2.5}$ (138)

Table 1.5: Summary of Exposure

Cities, Countries	Data from the Study: Theta (SE)	Conversion to PM_{10} per 10 $\mu g/m^3$ (SE)
Bangkok, Thailand	All ages – 0.104 (0.028) (3-day lag)	0.01 (0.003)
	>/= 65yrs - 0.10 (0.04) (3-day lag)	0.01 (0.004)
Santiago, Chile	All Ages - 0.075 (0.013) (1-day lag)	0.0075 (0.0013)
	>/= 65 yrs – 0.091 (0.017) (1-day lag)	0.0091 (0.0017)
New Delhi, India (TSP)	All Ages - 0.00023 (0.0001)/unit change in TSP (2-day lag)	0.0042 (0.002)
Mexico City, Mexico ($PM_{2.5}$)	All Ages - 1.36% (0.20 – 2.52%)/10 $\mu g/m^3$ change in $PM_{2.5}$ (current and 4-day lag)	0.0095 (0.004)
	>/= 65 yrs – 1.58% (0.04-3.12%)- (4-day lag)	0.0110 (0.005)
Sao Paolo, Brazil	>/= 65 yrs – 1.033 (1.006-1.060) RR for difference in 10^{th} – 90thcentile (1-day lag)	0.0051 (0.002)

A pooled estimate was drawn for the Bangkok, Mexico City and Santiago Studies which have both values for all ages and the elderly. The relative risks from the pooled estimates are as follows: for all ages =1.008 (1.006-1.010) and for the elderly relative risk = 1.010 (1.007-1.012).

A recent addition to the studies from developing countries is another time series study on Mexico City investigating the particulate effects on mortality as modified by socio-economic conditions using 1994-98 data. A 0.25% increase in total mortality was found to be associated with a 10 $\mu g/m^3$ increase in same day PM_{10}. Significant findings of the study included increasing strength of

associations between PM_{10} and total mortality with decreasingly advantageous socio-economic indicators especially for the elderly. Areas of residence were ranked into three categories: high/medium high, medium/medium low, and low/ very low. Among the elderly deaths, no association between mortality and PM_{10} was found for the high/medium high ranked areas. For the medium/medium low ranked areas, 0.68% increase in daily mortality was associated with 10 $\mu g/m^3$ change in PM_{10}. For the low/very low ranked areas, a 1.23% increase in daily mortality was found associated with 10 $\mu g/m^3$ increase in PM_{10}. For the all-ages mortality, no association was found at the two extreme categories but a positive association was seen in the medium/medium low ranked areas. The indicators used were the percentages of homes with piped water, homes with drainage, percentage of literacy and percentage of indigenous language speakers plus a composite index using all four indicators. Also, educational attainment and percentages of homes with electricity were assessed. Results from these latter two indicators were rather inconsistent, especially for percentage with electricity that did not exhibit any trend or significance. For educational attainment, the estimates were positive and significant only for the least educated (less than 3 years of schooling). Findings in the other categories were not significant although a trend exists when comparing the secondary (6-12 years of schooling), primary (3-6 years) educational level with the least educated. But it must be noted that those in the professional group exhibited positive estimates that were higher than the primary or secondary level estimates. However, this was not significant and was explained by the author as a consequence of high smoking prevalence among the professionals (*O'Neill et. al., 2000*). However, this latter comment by the author does not seem to be consistent with a time series study design since variables such as smoking are controlled for because they do not vary from day to day. Nevertheless, this study showed substantial effect modification by socio-economic factors in the association between mortality and PM_{10}. Being conducted in a city in a developing country makes it even more important and relevant for the purpose of this present assessment.

As far as usefulness of the studies is concerned, McMichael had argued that time series studies cannot ascertain the premature death due to air pollution beyond a few days (sometimes referred to as harvesting) (*McMicheal et. al., 1998*). This argument had been a source of controversy especially with regards the use

of time series studies coefficients in health risk assessment. (*Ostro et. al., 1998; Quenel et. al., 1999*) Recently however, this issue of harvesting has been addressed by Schwartz in an analysis of the Boston data (1979-1986) and by Zeger et al with the Philadelphia data (1974-1988). Shwartz had shown that by varying the time scales, the size of the effects vary as well. By moving the daily time series to a monthly time series, the deaths associated with increase of 10 $\mu g/m^3$ $PM_{2.5}$, increased from 2.1% to 3.75%. Time scales of up to 60 days were tested. Zeger et al on the other hand developed harvesting-resistant estimates by excluding the short-term information affected by harvesting. Both studies claimed that the size of the effects seen in the time series studies were actually consistent with the cohort studies (*Schwartz, 2000, Zeger et. al., 1999*). These studies have shown that the time scales for the association between mortality and PM_{10} may not be limited to a few days, however, its use for calculating long term effects of air pollution on mortality would still be limited.

Kunzli had examined this relationship between the particulate pollution - mortality time series studies and cohort studies quite clearly. The author cited four categories of air pollution-attributable deaths and then tried to define the role of the two types of studies according to such categories. The first category includes cases or persons whose underlying weaknesses in health were due to air pollution and the deaths were also triggered by the air pollution. The second category is similar to the first. However, the deaths were triggered by something other than air pollution. The third and fourth categories had cases with underlying weaknesses not related to air pollution. In the third category however, the deaths were triggered by air pollution while in the fourth, the deaths were facilitated by something other than air pollution. Hence the last category was not related to air pollution at all and could be dropped from the pollution-attributable deaths argument. From the three remaining categories, the cumulative effects of air pollution were apparent in the first two categories. In the third category, it was only the acute effect of air pollution. Time series studies capture those in the first and third categories and the cohort studies capture those in all three categories. The author also cited Schwartz's suggestion of increasing the exposure window up to 60 days but maintained that the effects seen would still fall under the short term category (*Kunzli et. al., 2001*). I agree with this view since the window of

exposure shown by Schwartz did not reflect a long term. As will be shown later, the long term effects are longer than the 60 days suggested by Schwartz.

Further conclusions from Kunzli's paper included the following:

a. The degree of anticipation of death in time series studies is not completely addressed since time is only indirectly estimated,
b. the years of life lost measure which is important in public health assessments such as the present study is only shown by the cohort studies,
c. for short term effects, time series studies should still be used,
d. attributable deaths in cohort estimates can be interpreted as additional deaths per year,
e. and lastly, that confounding and misclassification of exposure limits the validity of cohort studies.

In spite of the last conclusion, Kunzli recommended the use of coefficients from the cohort studies to calculate attributable risk for mortality for the reasons already stated. He acknowledged, though that further prospective cohort studies should be undertaken to confirm the present ones (*Ibid.*). These arguments, in my view, were convincing.

1.3.2. Ambient Air Pollution: Morbidity Studies

Morbidity studies regarding air pollution are reviewed here not for the purpose of estimating the impacts in this study but to provide a full picture of the mechanisms of effects of air pollution. As in the mortality studies, a few reviews of morbidity effects on air pollution have been done. Outcome variables investigated have been varied. Incident or prevalent cases of specific symptoms and diseases, changes in certain laboratory tests e.g. lung function test, frequency of utilization of health services, and hospital usage to restriction in activities, e.g. school absenteeism are the more common endpoints that have been studied with regards air pollution. In this section, some of these reviews and examples of specific studies are presented.

One such review is that on the Utah Valley studies done by Pope. Studies conducted in this area were at an advantage primarily due to the low smoking rates among the study population. From the various studies, Pope summarized that elevated PM_{10} levels generally result to changes in all the health endpoints investigated including decreases in lung function, increases in respiratory symptoms, non-malignant cardiopulmonary diseases and respiratory health hospital admissions. Although, Pope expressed concern over the limitations of the studies, he also pointed out that these limitations are quite common in other studies and that the results from the Utah Valley studies were largely consistent with other studies and coherent. (*Pope, 1996*).

Similarly, reviews by Dockery and Pope on both acute and chronic health effects of particulate matter found consistency and coherence among the different epidemiological studies regardless of areas or settings of the studies. Combined effect estimates for acute effects showed increases of about 0.8 to 1% in respiratory hospital admissions or emergency room visits per 10 $\mu g/m^3$ change in PM_{10}, 3% increase in lower respiratory illnesses and decrements of 0.15% in the forced expiratory volume (a lung function measure). Range of estimated effect measures due to chronic exposure were also computed for in the review. Respiratory symptoms and diseases, specifically emphysema, chronic bronchitis and chronic cough, were found to increase by 10-25% per 10 $\mu g/m^3$ increase in PM_{10}. Decreases in lung function were also found to be about 0-2% (*Pope et. al., 1997*).

Folinsbee, in his review of human health effects of air pollution, likewise documented not only health effects of particulate matter alone but also effects of other pollutants such as ozone, acidic pollutants, carbon monoxide, nitrous oxides, and lead. Several health effects were named including cardiovascular and neurobehavioral effects seen because of exposure to carbon monoxide and lead. Estimates from studies reviewed were cited, i.e. Utah schoolchildren: 3-6% decline in peak expiratory flow associated with levels of 150 $\mu g/m^3$ PM_{10} levels, since no attempt at pooled analysis was undertaken (*Folinsbee, 1992*).

A most comprehensive and thorough review and evaluation of the health effects of outdoor air pollution is provided by the State of the Art report of the

committee of the Environmental and Occupational Health Assembly of the American Thoracic Society. According to the report, short-term effects of high concentrations of particulate matter on respiratory morbidity and mortality are more or less proven. Furthermore, the report stated similar findings as that of Dockery and Pope with regards to associations of acute and chronic morbidity with levels of PM_{10} even at current ambient levels (30-150 $\mu g/m^3$) (*American Thoracic Society, 1996*).

In developing countries some morbidity studies have likewise been conducted. For example, a 29% change in chronic bronchitis for 100 $\mu g/m^3$ increase in TSP was documented in Shanghai. Respiratory symptoms and lung function variations were also investigated in other cities within China (*Xu et. al., 1994*). In Mexico City, 70% of morbidity cases of cough and phlegm production were attributed to an increase of 106 $\mu g/m^3$ in ozone (*Castillejos et. al., 1992*). Ostro (1994) and Brandon (1997) estimated that 10 million restricted activity days, 87 million respiratory symptom days and 3,580 premature deaths occur in Dhaka as a result of severe air pollution mainly contributed by motor vehicles.

The following are some selected examples of studies on morbidity effects of particulate air pollution, three of which were conducted among non-smokers and the fourth on children, thus the confounding effect of smoking is avoided.

Table 1.6: *Selected Morbidity Studies on Air Pollution*

YEAR	AUTHORS	LOCATION	FINDINGS/COMMENTS
1991	Abbey, Mills et al	California, USA	Seventh-day Adventists were monitored for 6 years and associations with long-term cumulative ambient air pollution were investigated. Increased relative risks for all female malignant neoplasms, respiratory symptoms and incidences of asthma, obstructive disease and chronic bronchitis associated with TSP levels. Confounding with smoking is clearly avoided in this population since most of the study subjects are non-smokers

Table 1.6: Selected Morbidity Studies on Air Pollution (Continued)

YEAR	AUTHORS	LOCATION	FINDINGS/COMMENTS
1993	Abbey et al	California, USA	Again non-smoker, seventh-day Adventists were the subjects. Statistically significant relationships were found between TSP and several respiratory disease outcomes. The importance of this study and the preceding is that smoking which is an important cause of respiratory diseases is avoided and the clarity of the relationship with air pollution is better seen.
1995	Abbey et al	California, USA	Similar subjects as the two preceding studies. This time PM_{10} was investigated and development of respiratory symptoms were the outcome variables. All outcomes had increased relative risks associated with PM_{10}. Attempts at cumulative expo-sure levels using hours/year of exposure to higher than 100µg/m^3 PM_{10} were done. This maybe a good measure of exposure and could be used in other studies.
1997	Braun-Fahrlander et al	Switzerland	Swiss schoolchildren were studied and symptoms of chronic cough, nocturnal dry cough, and bronchitis were positively associated w/ PM_{10}, NO_2,and SO2. PM_{10} has the highest relative risk at 3.07 between the most and the least polluted areas.

(Abbey et. al., 1991; Abbey et. al., 1993; Abbey et. al., 1995; Braun-Fahrlander et. al, 1997)

1.4. Biological Mechanism Underlying the Relationship between Particulate Air Pollution and Mortality

The nature of the particulate plays a most important role in the extent and character of the damage it causes the human body. The size or aerodynamic diameter of the particulate dictates the depth of its deposition within the respiratory system. The smaller the size of the particulate, the lower it is deposited in the lung parenchyma. Apart from size, other qualities of the particulate also give rise to the biological reactions seen in particulate deposition. Gwynn et al (*Gwynn et. al., 2000*) showed recently that acidic sulphate aerosols as component of PM pollution are highly associated with both daily respiratory hospital admissions and daily respiratory mortality. A strong acidic content is apparently a major factor. The heavy metals present such as lead, iron and copper contribute to the heavy metal load of the body potentially causing some toxicity, apart from possibly increasing inflammatory reactions in the lungs. Infectious agents that might have attached itself to the PM could exacerbate

existing pulmonary diseases. Together with the general chemical composition of the particulate itself, these are the most commonly believed characteristics of the particulate matter which elicit the biological reactions (*Utell et. al., 1997*). Although, a review of major and trace components of PM by Harrison and Yin concluded that the evidence does not support any single component of PM causing the adverse health effects (*Harrison et. al., 2000*).

Evidence from epidemiological studies had shown an association between particulate pollution and mortality. However, the mechanistic considerations for this observation have not been well defined. Nevertheless, recent studies have postulated and shown the probable mechanisms by which damage could occur. Seaton et al (*Seaton et. al., 1995*) suggested that ultra fine particles could instigate alveolar inflammation among vulnerable individuals. Such an occurrence exacerbates an existing lung disease as well as increases blood coagulability which in turn causes cardiovascular deaths among these vulnerable individuals in pollution episodes. This suggestion regarding the role of particle size has been looked into experimentally by several studies. These in-vitro studies have clearly shown that smaller particles could cause more damage than bigger sized particles. (*Murphy et. al., 1999; Monn, et. al., 1999*)

Experimentation carried out by Godelski et al on dogs shed further light on possible mechanisms of PM effects. The study showed that the cardiopulmonary effects such as decreases in heart and respiratory rates in relation to both cumulative and actual exposure, seen in non-compromised dogs are mediated primarily by the autonomic nervous system, an interplay between the sympathetic and parasympathetic systems, with some pulmonary inflammation. More significantly though, are the effects found on compromised dogs or dogs with coronary occlusion. These compromised dogs when exposed to the concentrated ambient particles showed ST segment elevation on ECG 1 minute sooner than controls and its magnitude was also increased. ST segment elevation on ECG is a sign of myocardial ischaemia. Thus, such an event could be life-threatening (*Godelski et. al., 2000*). A similar result was found in another study using healthy normotensive rats versus hypertensive rats (*Kodavanti et. al., 2000*). As described in these experiments, although cardiopulmonary effects such as variability in

heart and respiratory rates are seen in the normal animals, it is the most vulnerable or compromised, who are at most danger.

In humans, Peters et al also studied the hypothesis suggested by Seaton et al. The findings of this cross sectional study showed that plasma viscosity for both men and women were significantly increased during the days of pollution episode as compared to non-polluted days. The altered plasma viscosity due to inflammatory processes observed in the lungs was interpreted as a possible mechanism in mortality-air pollution relationship (*Peters et. al., 1997*).

As cardiac arrhythmia is another possible pathophysiologic mechanism especially in compromised individuals also suggested by Godleski et al in dogs, a few human studies have investigated this probable mechanism. Pope et al suggested, based on their study, that alterations in pulse rate may form part of the pathophysiology of the cardio-respiratory mortality due to particulate pollution (*Pope et., al., 1999*). Similar suggestions were proposed in two other studies which were investigating the cardiac autonomic response as evidenced by changes in heart rate and heart rate variability when exposed to particulate matter especially for those who are already compromised (*Gold et. al., 2000; Pope, et. al., 1999; Peter et. al., 2000*).

In summary, the plausible biological mechanism for mortality in particulate pollution relates primarily to pulmonary inflammation and release of mediators. These events lead to direct toxicity to the heart muscle or trigger autonomic responses, both of which could induce cardiac arrhythmia, fatal for those who are compromised, such as sick individuals and the elderly. These possible mechanisms are apparent in acute exposures to particulate pollution leading to death.

Effects of chronic exposures to particulate pollution have been extensively studied in population based cross-sectional studies where consistent results yielded association between mortality and particulate matter. The three cohort studies, Pope et al, Dockery et al and Abbey et al, had likewise found positive associations between particulate pollution and mortality. However, the biological mechanisms are not quite clear. Utell and Samet enumerated potential

mechanisms as air way and alveolar inflammation, which as discussed earlier, could lead to increased blood coagulability and other cardio respiratory responses, and increased susceptibility to infection due to impaired body defences (*Utell et. al., 1997*). With such sustained insult to the pulmonary system, lung function is likewise diminished. Evaluation of lung function with regards to particulate pollution has been carried out in a number of studies. The results, according to Pope and Dockery (*Pope et. al., 1996*), correspond to less than two percent decrease in lung function for every 10 $\mu g/m^3$ difference in exposure. Menkes et al, (*Menkes et. al., 1985; Menkes et. al., 1984*) in turn have found that lung function is a predictor of mortality. Lung function decreases after reaching its maximum performance at age 20-30 years old (*Evans et. al., 1997*). With a decreasing lung function performance due to age and a continuous exposure to particulate pollution all throughout one's life cycle, a combined effect can lead to life shortening.

Direct effects on the cardiovascular system may also contribute. Seaton hypothesised that the pulmonary inflammation caused by particulate pollution may have a role in causing atherosclerosis of the coronary arteries. With chronic exposure, low-grade chronic inflammation may result and the release of several inflammatory factors including fibrinogen could be continuous. Chronic increased levels of plasma fibrinogen, also a known coronary risk factor, may in due time lead to myocardial ischaemia. The fibrinogen infiltration of the blood vessel wall together with other processes such as platelet aggregation and increased blood viscosity explain the mechanism leading to this potentially fatal event. (*Torres et. al., 1996; Hernandez et. al., 2000; Montalescot et. al., 1998; McArty, 1999; Danesh et. al., 2000*)

To summarize, as Seaton suggested, inflammation due to particulate pollution releases fibrinogen which plays a role in atheroma formation. With constant and cumulative deposition and the infiltration of fibrinogen and other elements into the coronary blood vessels, myocardial ischaemia inevitably occur. Thus particulate pollution increases the chances of a constantly elevated plasma fibrinogen level due to the low-grade inflammation resulting from the chronic exposure.

1.5. The Environmental Health Risk Assessment Process

This thesis is exploring the potential for risk assessment to characterize environmental health impacts in complex social environments. This relies on applying risk coefficients derived from existing data to the population data in Metropolitan Manila. It would be desirable to estimate these coefficients directly in an epidemiological study in Metropolitan Manila but it is beyond the scope of the time and resources available in this project. Thus, the environmental health risk assessment process with some variations as models for this project would be tested.

Therefore, at this point, it is important to define environmental health risk assessment and describe its process. Risk assessment is defined as the quantification or characterization of potential adverse health effects due to human exposure to environmental hazards (*Griffith et. al., 1993; Smith et. al., 1995; Samet et. al., 1998*). This process has been evaluated in most detail in two slightly different attempts to structure risk assessment. These attempts are the USEPA model which is the most widely-used by risk assessors throughout the world and the Covello/Merkhofer model which treats the hazard identification step as an entirely separate process from risk assessment itself. Although different with regards to a couple of steps, the two models are essentially identical in concept and outcome. The process, whichever model it might be, is considered a tool for risk management used by both scientists and government officials alike.

In the USEPA method (*USAID, 1990*), the risk assessment process has four steps, namely hazard identification, dose-response assessment, exposure assessment and risk characterization. Each step answers a different question relevant to a final estimation of the risk among the exposed population. The hazard identification step documents the health problems caused by a certain pollutant. This step considers scientific appropriateness of the evidence with regards to inferences from one setting to another, e.g. from animal experiments to humans. Carcinogenic and non-carcinogenic effects are dealt with separately. The USEPA has developed a formal procedure for weighing toxicological and epidemiological studies for carcinogenic substances. The procedure results in categorizing the pollutant in terms of its carcinogenic potential. For the non-carcinogenic health effects, the process is more qualitative in approach. Studies

are assessed according to a set of criteria and then a subjective summary judgment as whether the pollutant causes a particular health effect is made (*Ibid.*).

The dose-response assessment makes use of published data correlating exposure levels with adverse health effects. This step involves estimating quantitatively the relationship between the amount of exposure to a pollutant and the likelihood of a health effect. Two types of effects are distinguished in this process, the threshold and non-threshold effects. For non-threshold effects, extrapolation of the exposure range data to human environmental exposures may be necessary, especially in carcinogen risk assessment. A linear relation (down to zero) between the dose and response is usually assumed for estimating cancer risk. For threshold effects that apply mostly to systemic pollutants and non-carcinogenic health effects, calculation of an acceptable daily intake value or reference dose is done. Uncertainty or safety factors are also incorporated into this reference dose for this level to be more likely below the lowest observed adverse effect level.

Exposure assessment identifies exposed populations, and the specific agents, and evaluates the extent, duration and amount of exposures. This step also involves, source assessment, fate analysis of the pollutant in the environment, and estimation of environmental concentration. The result of this step is then incorporated into an integrated exposure analysis that also evaluates the uncertainty.

Risk characterization is the last step in the process and integrates the hazard identification, dose-response assessment and exposure assessment. The outputs of the process refer to estimated risks to individuals as well as to populations.

GENERAL STEPS IN RISK ASSESSMENT

USEPA model:	Covello/Merkhofer model:
• Hazard Identification	• Release Assessment
• Exposure Assessment	• Exposure Assessment
• Dose-response Assessment	• Consequence Assessment
• Risk Characterization	• Risk Estimation

In the Covello/Merkhofer model (*Covello et. al., 1993*), the process still retains the number of steps. However, the first step here is the release assessment that includes a description and quantification of the potential of a risk source to release specific hazardous agents. It is presumed in this model that the hazard identification step has been done separately. Exposure assessment is next, similar to the USEPA model, followed by the consequence assessment. This latter step actually approximates the dose-response assessment step in the other model since it also describes and quantifies the relationship between exposure and effect. Finally, risk estimation is done wherein results of the first three steps are integrated into a quantifiable measure of risk. Krzyzanowski recommends this model as a method for assessing the extent of exposure and effect of air pollution (*Krzyzanowski, 1997*).

Both models could make use of animal and human epidemiological studies. However, it is important to note that, although, the use of epidemiological studies is fairly recent in risk assessment, the advantages of doing so are considerable and it has been argued that its use must be encouraged (*Samet et. al., 1998; Hertz-Piccioto, 1995*).

Among the advantages mentioned, the concept that no interspecies extrapolation is needed for both hazard identification and dose-response assessment is the most essential justification for using epidemiologic research for risk assessment. It was further pointed out that using animal data would lead to a magnitude of error far greater than when human data is used. Other advantages indicated are the need for a smaller range of extrapolation, the fact that exposure scenarios in animal studies are poor representations of human exposures and that the heterogeneity of populations being assessed is better represented by human studies. Although human data have also their limitations such as undetected potential confounding, problems with validity of exposure and outcome measurements and study size, to name a few, the advantages far outweigh such limitations (*Smith et. al., 1995; Hetz-Picciotto, 1995*).

Having said that, it is most important to assess epidemiological studies according to its acceptability of utilization in environmental health risk assessment. Criteria have been set with regards to this, which is not exactly all

that different in terms of assessing causality in epidemiological studies. Both the qualitative and quantitative nature of the epidemiological studies is taken into consideration. In the classification of epidemiological studies framework proposed by Hertz Piccioto for example, studies are categorized according to the following criteria: its use in risk assessment, strength of associations found, control of potential confounding, ruling out of possible biases, quantification of exposures linked to individuals and the presence of monotonic dose-response relationship (*Smith et. al., 1995; Samet et. al., 1998*).

In air pollution health risk assessment, Ostro has named three factors that must be addressed. The following summarizes these three factors. The first is the development of dose-response relationships involving careful selection of studies. Several criteria for the selection of studies are enumerated. These criteria are quite similar to that proposed by Hertz-Piccioto but more specific to air pollution studies i.e. selected time series studies must control for seasonality and weather and has to provide particulate measure which could be converted to PM_{10}. From these studies, an estimate of the change in the health effect probability given a change in the pollutant with all other factors held constant, is established. The second factor is determining the exposed population. In air pollution risk assessment, this would almost always include the entire metropolitan area population. Exposures could be ascertained by fixed-site monitors or determined from dispersion models. For certain specific health-related outcomes such as asthma attacks, sensitive populations may be identified as well. The third factor is the change in air pollution under consideration or the alternative exposure scenarios. A target concentration from which the current ambient air pollution level is ascertained. For this purpose, the air quality guidelines or standards may be used as the desired target concentration (*Ostro, 1996*). The assumptions and other aspects of the three factors mentioned will be more thoroughly discussed in the methodology section.

In environmental health risk assessments done in some countries in Southeast Asia (*USAID, 1990; WB, 1996*), it is the USEPA model that was used. The USEPA model will be referred to for this project since the difference between the two models is almost negligible and for the purpose of comparison later on.

It is also relevant at this point to present the concept of health impact assessment as opposed to health risk assessment. In a report submitted by Frankish, et al to the Health Promotion Development Division of Health Canada in 1996, health impact assessment was defined as any combination of procedures or methods by which proposed programmes may be judged as to the effect(s) it may have on the health of a population. It implied the quantification of expected health outcomes due to specific exposures in certain populations.

Health impact assessment is one of two distinct components in health risk assessment, the other being health hazard characterization. Health impact assessment reflects the interplay of the exposure assessment, dose-response assessment and risk characterization in the USEPA model previously discussed (*WHO, 2000*).

Locally, the terms impact assessment and risk assessments are used interchangeably. Some implementers use the term health impact assessment in their work, but strictly following the content and process, health risk assessment is actually being done.

1.6. Environmental Health Risk Assessment in South East Asia and Other Regions

There are two particularly interesting environmental health risk assessments done in Southeast Asia, the Bangkok study by the USAID, USEPA in 1990 and the Philippine study by the World Bank in 1996. Another assessment was also conducted in Jakarta, Indonesia by Ostro, also for World Bank in 1994, particularly for air pollution.

The Bangkok assessment objectives included the determination of whether the USEPA methodology, described in a preceding section, could be adapted for application in the developing world and to establish and compare the different urban environmental health risks in Bangkok. The methodology for the study was known as 'comparative risk analysis'.

All aspects of environmental pollution were assessed. Variations to the USEPA method were also made; one of which is relevant to this project and

pertains particularly to particulate air pollution. This variation involved the inclusion of epidemiological studies establishing the relationship between air pollution and health. From these studies, estimates of excess mortality and restricted activity days were calculated. For the whole assessment, population risks were estimated and no estimates were calculated for specific vulnerable groups (*USAID, 1990*). Population risk estimates are extremely useful for policies e.g. setting-up of emissions and ambient air standards, decreasing pollutants in the ambient air by limiting transport vehicles or decreasing the number of industries operating.

A more recent health assessment was made by Chestnut et al which covered premature mortality hospital admissions and respiratory symptom days. The assessment yielded a 52 – 87 deaths per million population attributable to 10 ug/m^3 increase in PM_{10}. The exposure response coefficients used for this assessment were based on local daily time series studies. (*Chestnut et al, 1997*) However, the assessment did not include long term mortality attributable to air pollution.

The objective of the Jakarta study was primarily to 'describe a method for determining quantitative estimates of the benefits of reducing ambient concentrations of particulate matter, sulphur dioxide, nitrogen dioxide, ozone and lead'. The method used in this study was, again, the USEPA method with dose-response functions taken from the more recent studies and with more health endpoints included in the estimations. The entire population was generally considered as the exposed population except for specific health endpoints where children and asthmatics were singled out (*Ostro, 1994*). Attempts to distinguish the effects of air pollution on the more vulnerable groups of the population were limited to estimating risks for those with specific medical conditions and younger age groups.

The Philippine study on environmental health risks involved estimations of health effects of different environmental pollution for the whole country. Five principal environmental exposure pathways (PEEPs) were identified and the burden of mortality and morbidity were estimated. The PEEPs defined the ways by which populations are exposed to the pollutants and tried to identify the

different vulnerable groups, especially in the air pollution section. For the burden of morbidity and mortality, the days of healthy life loss was used as a measure of the health outcome. The dose-response coefficients used were based on cross-sectional epidemiological studies. For air pollution, estimation of the morbidity burden for certain susceptible groups was done e.g. schoolchildren and jeepney and bus drivers. This estimation was possible because of the presence of measurement data from local epidemiological studies for those particular groups (*WB, 1996*). Although, this study estimated the burden of disease for some specific vulnerable groups such as schoolchildren and jeepney drivers, due to air pollution risks, it was rather specific and did not address the heterogeneity of the population in general, thus not providing comparative risks within the population. Also, mortality due to air pollution for these vulnerable groups was not assessed. Since the dose-response coefficients used were based on cross-sectional studies, the interpretation of days of health life loss and even the premature mortality estimated to be due to air pollution is problematic. Nevertheless, estimations done in this study were extremely useful for pushing the Clean Air Programme of Metropolitan Manila but health programmes geared towards such air pollution risks are still wanting.

Similar environmental health risk approach has been used in estimating premature mortality in other cities in Asia and in Europe. In Dhaka, Bangladesh, it was estimated in 1994 and in 1997 by Ostro and Brandon respectively, that 3,580 premature deaths could be attributable to air pollution. However, the method used for estimation was not evident from the report (*Dhaka study, 1998*). In New Delhi, India, Cropper et al tried to estimate the premature deaths and the years of life lost attributable to TSP based on the exposure–response coefficients calculated from the New Delhi time series study, comparing it with using exposure-response coefficients from Schwartz and Dockery Philadelphia time series studies. The method of estimation is similar to that used in the studies (Philippines and Jakarta, Indonesia) earlier discussed. The comparison yielded higher number of deaths but lesser years of life lost when using the Schwartz and Dockery coefficients as opposed to using the New Delhi coefficients which yielded lesser number of deaths but higher number of years of life lost. These results, as explained by the authors, were most probably because there were more deaths at younger age groups in Delhi than in Philadelphia and the impact

of air pollution on deaths is greater at younger age groups in Delhi than in Philadelphia. Thus, applying the US time series exposure-response coefficients in populations exposed to higher pollution levels for risk assessment was deemed questionable (*Cropper et al, 1997*). However, as discussed in section 1.3.1, the Delhi time series study data set is quite problematic in its completeness, as admitted by the authors, so as to derive the conclusion about effects in different age groups.

Three important European public health assessment studies of air pollution have been done by Kunzli et al, Brunekreef and Hurley et al in Austria-France-Switzerland, Netherlands and the United Kingdom respectively.

In the Netherlands assessment by Brunekreef of the effect of particulate pollution on long term mortality using the life table approach and the exposure response function estimates based on the cohort studies of Pope and Dockery, a difference of 1.11 years in life expectancy among males living in clean air as compared to those exposed to dirtier air was seen. The exposure response function estimate used in this study was quite high at 1.10 relative risk per 10 ug/m^3 PM_{10} increase (*Brunekreef, 1997*). Thus, the effect seen on the life expectancy was also a longer period of time lost.

The assessment done by Hurley et al for the United Kingdom was more extensive with regards the health outcome variables used and economic analysis as well. In this Hurley et al study, effects on acute and chronic mortality and respiratory morbidity including hospital admissions and restricted activity days were assessed (*Hurley et al, 2000*). Similar assessment was done by Kunzli et al in three European countries mentioned earlier. Several outcome variables were likewise considered including long term mortality using the estimates from the ACS cohort study by Pope et al. Six percent of the total mortality per 10 ug/m^3 increase in particulate pollution in the three countries were considered attributable to air pollution (*Kunzli et al, 2000*). Although Kunzli et al made use of the cohort study exposure response coefficients by Pope et al, the years of life lost were not estimated unlike in the Hurley et al study. These aforementioned health assessment studies are very important references for this study.

As seen from these examples of risk assessment, the USEPA method could be adapted, in spite of uncertainties especially with regards to the calculation of the absolute and reliable estimates of the risks and the burden of disease. However, further modifications that consider the heterogeneity of the population, as exemplified by difference in socio-economic status, can be made. The rationale for such modifications and the modifications themselves are the topic of this project and are discussed in the next sections.

1.7. Vulnerable/Susceptible Groups, Air Pollution and Health

Vulnerability in the Oxford dictionary and Thesaurus also means susceptibility (*Oxford University Press, 1997*). For the purpose of this project, these two words are used interchangeably. Parkin and Balbus have reviewed variations in the concept of susceptibility in risk assessment. They have described discipline specific and interdisciplinary definitions of susceptibility. Consequently, the emphasis of each definition depends on the underlying perspectives and methods of each field. Due to the variations in definitions, it was their recommendation that for the purpose of any risk assessment, the assessors must state explicitly beforehand the definition of this term (*Parkin et. al., 2000*).

The US Clean Air Act requires that sensitive subpopulations should be considered in setting standards. Similar to the definitions of vulnerable groups in health studies, the list of these sensitive subpopulations encompass the very young, the very old and persons with pre-existing disease or disability. Understandably so, these subgroups, due to their physiological make-up or intrinsic characteristics, are most sensitive to insults brought upon by environmental pollution. However, aside from the differences in vulnerability because of age and other intrinsic factors such as genetics, extrinsic factors also play a role in determining vulnerability/susceptibility. These extrinsic factors may include place of residence, community level of exposure, poverty, health care access, occupation and or/ education (*Parkin et. al., 2000*). The Philippine Clean Air Act has a similar mandate as that of the US (*EMB-DENR, 2000*). To this end, it is very important for this risk assessment to describe the public health impact in terms of some of these sensitive subpopulations. With regard susceptibility to air pollution according age, as discussed in the epidemiologic evidence section, studies have shown differential mortality effects of air pollution among the elderly

as well as independent mortality effects among the infants. Hence, age must be considered. Apart from age, it is also the intention of this project to assess the mortality impact of air pollution according to socio-economic classes, especially the urban poor, described earlier as one of the extrinsic factors in susceptibility.

The vulnerability of the urban poor is rooted in the fact that together with the high prevalence of malnutrition and infectious diseases, they are also exposed to environmental insults and other 'benefits' of development for which they may or may not be more sensitive. Thus, in doing health risk assessments in cities of the developing world, it is essential to consider these vulnerable subgroups and their patterns of exposures.

In the ensuing topics, differences in health among different socio-economic classes are discussed briefly as well as the different indicators used for measuring the said variable. This section would show that differences in socio-economic status are an important determinant of health. In addition, a few studies would be presented regarding the health effects of environmental pollution, in this case air pollution, as modified by the effects of socio-economic status. Thus, the importance of considering the impact of certain exposures with regards to different socio-economic classes, a marker of population heterogeneity, is underlined.

1.7.1. Vulnerable Groups and Socio-economic Status

Socio-economic status of individuals or populations is regularly measured in epidemiological studies whether for the purpose of comparison across social classes or is treated as a confounder. However, such studies, depending on the country, have different ways of measuring socio-economic status. This section will review the different definitions of socio-economic status used in several mortality studies and will present the practicality of using such definitions.

The most common indicators of socio-economic status used in mortality differential studies are education, employment status, housing tenure, crowding, occupational class, place of residence and household income. Other less commonly used parameters are ethnicity, percentage of income spent on food, and possession of certain consumer durables or the material standard of living.

In the developed world, such measurements are relatively easy to obtain from records like the national census office or the health department, or from surveys conducted regularly (*Sundquist et. al., 1997; Kunst et. al., 1998; O'Shea, 1997; Sastry, 1997; Mustard et. al., 1997; Duncan et. al., 1995; Songsore et. al., 1993*). Such socio-economic indicators are regularly collected in national census surveys normally done every ten years or may be available in the death certificate data. To a certain extent, such data are also available in developing countries but with further limitations in completeness and validity

The level of educational attainment is the most commonly used socio-economic parameter in many epidemiological studies (*Liberatos et. al., 1988*). This is defined as the number of years of schooling completed or classified further as low, intermediate and high (*Sundquist et. al., 1997*), grade school, high school, college, certificate and higher education (*Mustard et. al., 1997*). Education is said to be the most stable indicator since it does not usually change when a person reaches adulthood and remains unaffected by poor health later in life (*Ibid.*). Kitagawa and Hauser also mentioned that education as an index can be defined for both economically active and inactive persons in the population (*Kitagawa et. al., 1973*). Education's strong association with population health was found in a study by Jerrett among the populations in 49 counties in Ontario, Canada where it turned out to be the strongest predictor of premature mortality especially among the females (*Jerret et. al., 1998*). However, as pointed out by Valkonen, in Finland and in some parts of Europe, level of education may not be as useful an indicator in mortality differential studies as in the United States or Canada. Valkonen commented that since everybody has the same level of education as prescribed by compulsory schooling, the distribution by schooling years will be skewed, thus, making the analyses of differences in mortality more difficult because the level of education would not vary sufficiently (*Valkonen, 1993*).

More commonly used indicators in Europe are the occupational class and employment status. In the United Kingdom, social class is primarily based on occupation as reflected in the British Registrar General Classification. Other schemes of classification are the EGP social class scheme and the Wright Scheme, based on job title, employment status and supervisory status (*Kunst, 1997*). Although these latter schemes have the advantage of being more

comprehensive, the collection of such data maybe a little more difficult. Classification of the economically inactive part of the population may also be problematic. An advantage may be that occupational classifications also reflect exposures experienced by individuals, to a certain extent, which may be useful in helping to identify probable causes in mortality studies. However, like income, occupation may change throughout the lifetime of an individual making it less stable than education as a socio-economic indicator, though it may better reflect those changing circumstances that affect health.

Income is another indicator that is quite difficult to collect in surveys. People are usually hesitant to divulge their income for a variety of reasons. Nevertheless, income, occupation and education are considered in many studies as the core indicators of socio-economic status (*Ibid.*).

Overcrowding, measured as the number of persons per bedroom or space per person in a house, and the possession of consumer durables in the house are also useful indicators not only in the developed world as used by Weich et al in Britain (*Ibid.; Weich et. al., 1998*) but also in developing countries. In fact, epidemiological studies in the Philippines have used these two parameters more often than just household income or education. These two parameters are easy to collect and handle in surveys. However, although useful in rural areas, socio-economic scoring according to the number of consumer durables such as television, stereo sets and the like, may sometimes fail to discriminate in urban poor areas like in Metro Manila. This is because even in squatter areas, such consumer durables are almost always present, no matter how poor a family is, and therefore may not truly reflect the socio-economic status of a particular household.

Percentage of income spent on food is another measure of SES used commonly in comparing large geographic areas such as districts, cities, (*Songsore et. al., 1993*) countries and even regions. But such measure may not be as practical when comparing individuals in the lower classes because it will not delineate them sufficiently.

In countries and cities, which are very mixed in terms of ethnic groups or are very cosmopolitan such as those in the United States, ethnicity has been used quite often as an indicator that correlates to some extent with socio-economic status. Racial differences have been frequently regarded, unjustly though it may seem, as predictor of socio-economic class. In developing countries, especially in those areas where the population remains homogenous, such ethnic comparisons cannot be employed.

Not surprisingly, people with similar cultural and socio-economic background tend to mingle amongst themselves and live near each other. Thus, geographic location or place of residence as unit of analysis for investigating socio-economic status could be used. Easily recorded in death certificates, the place of residence indicator is practicable. The main drawback of its use as an indicator however, is the lack of information in the length of time that a person has resided in such an area. Knowing the time period will help in determining lifetime exposure to such standard of living that in turn may delineate more clearly the effect of socio-economic status.

The more practical way by which socio-economic status is represented in many studies, short of an application of a composite index, is to consider a few indicators at the same time. This is most common in mortality differential studies as well as other epidemiological studies. However, correlation amongst the different indicators must be explored in order to assess the practicability of using such combination of indicators. As mentioned earlier, the core indicators are occupation, education and income. Each of the said core indicators can pinpoint different aspects within public health policy that could be changed (*Kunst, 1997*).

Another set of indicators is the material standard of living wherein several factors are taken into consideration. These are savings from income, access to a car/van within the household, number of domestic household appliances, housing tenure, overcrowding and presence and number of structural housing problems (*Kitagawa, 1973*). More of such combinations will be discussed in the next part. Many other combinations are also prevalent in the literature. However, regardless of the indicator used in health differential studies, the results from all

these studies indicate that populations in the lower socio-economic class are almost always disadvantaged with regards to their health.

1.7.1.1. Composite Indices - Advantages and Disadvantages, Examples

Variables that are grouped together and given different weights to represent a specific, single entity could be considered a composite index. In determining socio-economic status of an individual or a population, several types of indices have been formulated. Examples abound in the literature such as the more popular poverty index, used generally by countries as a measure of population living below the 'poverty line'. The 'household wealth index' as expressed by Songsore in his study in Ghana, is measured in terms of possession of certain consumer durables and frequency of consumption of meat, poultry and fish. Using this index allowed Songsore to create wealth quintiles for analysis of environmental and health problems (*Songsore et. al., 1993*). Commonly used in the United Kingdom is the deprivation index as opposed to the poverty index. It is important to know the difference between deprivation and poverty. Townsend in his paper on deprivation pointed out that people could experience one or more forms of deprivation without necessarily being in poverty. Deprivation, as a concept, encompasses a broader view. Included in such a concept is not only the material deprivation but also social. Thus whereas poverty is confined to the physical lack of amenities, deprivation would also look at the lack of opportunities to participate in family, community and other recreational and educational activities (*Townsend*). However, social deprivation is quite difficult to measure and would need a more complicated method of assessment. Thus, most studies confine themselves to material deprivation.

There are at least five well-established deprivation indexes used in the UK. These are the TOWN index by Townsend et al, the SCOTDEP by Carstairs, JAR by Jarman, DOE by the Department of Environment and the SDD by the Scottish Development Department. Morris and Carstairs present a comparative summary of the variables used in the different indexes as well as the processing of these variables. Unemployment is the most common variable used in all fives indexes. SCOTDEP authored by Carstairs herself uses only four variables, which includes access to a car, social class status, and overcrowding, while the other indices have at least five (TOWN) to about eleven (DOE). The study found that

among the five indexes, SCOTDEP and TOWN could explain most of the variation of material deprivation in relation to health and mortality. These two indexes also adhere most closely to the concept of material deprivation (*Carstairs, 1995; Morris et. al, 1991; Jarman, 1983*).

Stephens in her thesis on mortality differentials in Sao Paolo, Brazil came up with a composite index using socio-economic and environmental variables (*Stephens, 1999*). Such a combination of variables is useful in the mega cities of developing countries where access to certain environmental utilities e.g. sanitation and waste disposal facilities, may also be reflective of the socio-economic status of the different populations living in such cities. However, the problem with such an index, and with most composite indexes, is that it fails to delineate individual effects of the variables from the effects of their interaction. This is a disadvantage inherent in any simplified single index (*Kolsky et. al., 1995*).

In addition, although composite indexes are quite useful as a tool and simple enough for public health policy-makers, so long as they understand what it means, it is usually only relevant to the place/city/country where they are developed. An index in one country may not be functional for another, considering the differences in development, culture and even values. Thus, international comparisons using only one particular index will be impractical. This is the main difficulty of formulating a composite index or a single indicator for international use.

1.7.1.2. Social Inequality Indices in Mortality Studies

Aside from composite indexes used to measure socio-economic status, several other indexes are also formulated to measure not just the socio-economic status of different populations but the inequality that may exist among such populations. Kunst in his book on the Cross-national comparison of socio-economic differences in mortality summarized 12 most used indices. Kunst divided the indices into the effect indices and the indices of total impact. The effect indices include the mortality rate ratio and rate difference of the disadvantaged versus the advantaged groups and the regression-based index of relative effect and absolute effect. The indices of total impact are the population-attributable risk (% and absolute), regression-based population attributable risk

(% and absolute), the index of dissimilarity (% and absolute) and the regression-based index of dissimilarity (% and absolute) (*Kunst, 1997*). Advantages, disadvantages and uses of these indices are further enumerated by Kunst and can be referred to in his book. It is important to note though from Kunst's discussion, that there is no perfect index and the use of a particular index depends primarily on the intention or purpose of the user/researcher.

In another study, Kawachi et al tested the relationships among the six different income inequality indicators to the total mortality rates in 50 U.S. states. The six summary indicators of income distribution were the Gini coefficient; the decile ratio; the proportions of total income earned by the bottom 50%, 60% and 70% of the households; the robinhood index; the Atkinson index and the Theil's entropy measure. Besides being highly related to mortality, all six indices were found to be strongly correlated with each other as well. Thus, it was concluded by the study that the choice of inequality index did not affect the relationship between inequality and mortality (*Kawachi et. al., 1997*).

Most of the studies cited using such inequality indices were done in developed countries (*Kahn et. al., 1998; Kennedy et. al., 1996*). It will be interesting to use these indices in mortality studies in developing countries where the difference between the rich and the poor is much greater. These inequality indices are quite restrictive in a sense, because it only reflects income. Also, other aspects of deprivation are not considered, which may well be more important in assessing standards of living. Furthermore, as surmised earlier in the discussion, and also according to Wilkinson, the evidence for the association between income inequality and mortality is sensitive to the response rates to income surveys. This means that the more equitable the income distribution is in a country, the lower the response rate. Therefore, non-correction for this non-response bias could affect any association seen. In the Philippines, response to household income surveys are not generally poor, however, it is also observed that underestimation of income by respondents in surveys is prevalent, leading to inaccurate conclusions.

As revealed by this brief discussion on the effects of socio-economic status and inequality on health, it is important to assess health according to such

variable. Thus, it is equally important that health impact of any environmental pollution must also be assessed accordingly as the effects may vary. This latter will be addressed upon in the next section.

1.7.1.3. Studies Regarding Modifications of Effects of Air Pollution on Health by Socio-Economic Status

Although many studies have investigated mortality differentials among different social classes, studies dealing with effect modifications of socio-economic status on air pollution effects on morbidity and mortality are quite scarce. Most studies that had come out in the past two decades have in general controlled for socio-economic status either in their design i.e. time series studies, or in their analysis, i.e. cross sectional studies. Salinas et al in Chile had results that imply that deaths due to COPD and asthma are associated with chronic exposure to heavy air pollution and not to deteriorating living conditions (*Salinas et. al., 1995*). However, in addition to an unclear definition of deteriorating living conditions, there were a few limitations, namely the lack of air quality data and the study design itself, which rendered the conclusion questionable. Furthermore, the effects of the deteriorating living conditions on the air pollution health effects were not elaborated on. In the thesis of Gouveia on the time series analysis of air pollution and health effects in Sao Paulo, Brazil, he found a slightly higher relative risk, though not statistically significant, for individuals who lived in the more wealthy areas as compared to those in the deprived areas (*Gouveia, 1997*). This finding is interesting since it is generally believed that the more deprived individuals exhibit a higher relative risk.

A review of papers on air pollution health risks and the relevance of class and race by Sexton et al, revealed a few interesting studies. Most significant in the review was a study by Gelobter, which found that different socio-economic and racial groups are exposed differentially to outdoor air pollution. However, no associations with health outcomes were investigated (*Sexton et. al., 1993*). This review, nevertheless, acknowledged that more studies on the topic are needed. Another study which could be interesting is that done recently by Rotko et al which documented personal exposure to fine particles ($PM_{2.5}$) in Expolis, Helsinki. In that study of 25-55 year old inhabitants of Metropolitan Helsinki from October 1996 to December 1997, the authors had found that lower occupational status, less educated and young participants had more exposure to air pollutants than

the upper occupational, more educated and older participants. However, this socio-economic difference was explained mostly by the difference in workplace concentrations rather than outdoor concentrations of pollutants (*Rotko et. al., 2000*). In another study conducted by the Environmental Epidemiology unit at London School of Hygiene and Tropical Medicine in 1997, associations between the air pollutants, socio-economic status as measured by the Carstairs index, and health outcomes were found to be inconsistent (*SAGE Project Group, 1998*).

In the study conducted by Sainsbury et al wherein the results exhibited the correlation between deprivation and mortality was stronger than the correlation between atmospheric pollution, measured in terms of industrial land use, and mortality. Nevertheless, the study also showed higher mortality among the most deprived in the most polluted areas as compared to those in the intermediate pollution category, but not in the least polluted. The results of this study led Sainsbury to speculate that among older people, the adverse effects of air pollution is seen only in the less deprived because in the most deprived individuals, the deprivation itself overwhelms the effects of air pollution. This could only mean that other health risks associated with deprivation might have come into play. However in younger people (<65 years), the adverse effects of air pollution are seen only among the most deprived perhaps because their health has already begun to be compromised (*Sainsbury et. al., 1996*). These findings and commentary suggest an effect modification of deprivation as well as age on the health effects of atmospheric pollution.

Recent studies, however, have started investigating the role of social status as modifier on the effect of air pollution on mortality. Zanobetti and Shwartz analysed the total daily mortality of four cities in the US with the daily measurements of PM_{10}. Their findings showed some effect modification of race and educational attainment, but quite weak and modest compared to the effect modification by medical conditions (*Zanobetti et. al., 2000*). However, in a time series study done in Mexico City, significant effect modifications by several socio-economic indicators were found. (*O'Neill, 2000*) Further more, the recent reanalysis of the cohort studies have also shown such an occurrence using education attainment as the socio-economic indicator (*Krewski et. al., 2000*). These latter two studies are discussed more extensively in Chapter 3. Hence, evidence

of differential air pollution effects on mortality by socio-economic status is accumulating.

Another aspect that could be explored with regards to effect modification is the effect of air pollution on those who are chronically sick such as asthmatics, and those with chronic obstructive disease or bronchitis as evident in some studies (*Brunekreef et. al., 1993; Romieu et. al., 1996; Ponka et. al., 1994*). If these susceptible subgroups are more prevalent among the lower socio-economic classes, these classes are then made more vulnerable. Health studies conducted in Metropolitan Manila indicate such occurrence (*WB, 1996*).

As seen from this review, there is evidence of health differentials between socio-economic classes. Also, some evidence of differential exposures to air pollution of different social classes, as well as differences in health impact of environmental pollution exists. In addition, a couple of time series studies, one of which was undertaken in a developing country, and the cohort studies on mortality and air pollution have documented variations in effects of air pollution by socio-economic status.

1.8. Conclusions on and Limitations of the Current Model of Environmental Health Risk Assessment

There is little doubt that air pollution could affect health as seen from this review. Age and socio-economic status interactions with air pollution have also been shown in some studies. Thus, it could be inferred that differential health effects of air pollution on people with respect to these two variables could also result.

The USEPA method of health risk assessment in general and the air pollution health risk assessment in particular, are good models that have been used extensively in developed countries. This method could also be potentially adapted to cities in developing countries as exemplified by the Bangkok and Philippine assessment studies but with a few variations. However, such approaches in the assessment of the burden of disease attributable to environmental pollution, have not really distinguished between the most and least affected parts of the population, even in the Bangkok and Philippines studies.

This aspect is extremely important in cities of the developing world. The heterogeneity of populations as measured, for example, by socio-economic classes, is an aspect that has not been detected in the health risk assessments done. Hence, the tendency to over- or underestimate disease burden in different subgroups of the population, as the case may be, is an apparent possibility. It is for this reason and the need to redefine vulnerable groups that an appropriate approach to risk assessment must be explored, especially in the context of developing cities/countries.

In addition, the Years of life lost and loss in life expectancy measures are useful indices in estimating the burden of diseases. They are practical, and at present, used in several countries for cost-effective analysis and in health planning. Hence, these measures must also be explored with regards to its use in environmental health risk assessment.

The following sections will describe the rationale, hypothesis and objectives of the study. Furthermore, a detailed methodology by which this study will be conducted is outlined in the next chapter. This is followed by a description of the succeeding chapters.

1.9. Rationale of the Study

Although, the USEPA method could be used to investigate distribution of risk issues in different vulnerable groups (especially pertaining to lower socio-economic classes) within a population, this has not been done satisfactorily in the risk assessments made so far, including those in Southeast Asia. However, looking at these risk distributions in different subgroups of populations is of importance for three reasons. First of all, it could be beneficial in focusing public health programmes with maximum health gains for these particular subgroups. Programmers may be able to plan for specific target populations and be able to assess the benefits more efficiently. In addition, due to some detail that this kind of assessment will provide, policy responses may be more focused. Secondly, a more accurate picture of the burden of disease from particular risk factors may be provided. Enquiring into the estimates of different components of a whole could give more precise answers than analyzing the whole alone. Thus, more reliable answers as to the number of people affected and who they are, are given.

Thirdly, taking care of the more vulnerable groups in the population is a moral obligation of any government. This last issue is emphasized quite prominently by the World Health Organization that has as a major target - 'Equity in Health' (*Whitehead, 1998*). The only way to work towards this target is to know exactly what is going on in these different vulnerable groups.

Quite importantly as well, although an assessment exercise has been done for the Philippines as a whole as mentioned earlier, specific components in risk assessment used here are different than that used previously, e.g. dose-response coefficients based on cohort studies and the life table approach. These specific components, as will be discussed later, could render the estimates drawn to be more reliable and easier to interpret than the previous assessment.

1.10. Purpose and Objectives

Purpose of the study

To assess the potential magnitude of environmental health inequalities in a Mega-city in a developing country with particular reference to particulate air pollution in Metropolitan Manila.

Objectives of the study:

1. To estimate the chronic health impact of annual levels of particulate matter using routinely collected mortality, pollution, and census data.
2. To adapt risk assessment methods
3. To estimate the effect modifying impact of age and socio-economic status on effect estimates of links between long-term exposure to particulate matter and health.
4. To estimate the impact of various pollution reduction scenarios on effect estimates

In Chapter 2, the method by which the preceding objectives of the study were carried out is described. A general picture of the characteristics of Metropolitan Manila, the sources of data and procedures of data collection are included. In addition, a description of the analytical methods is presented.

The results are divided into three chapters. Chapter 3 describes the results of coming up with the appropriate exposure-response functions to use for the estimation. This chapter includes systematic reviews of the studies use and an assessment of the exposure of the population of Metropolitan Manila including the introduction of the pollution reduction scenarios. Finally, it outlines the main findings of the risk assessment. It presents the different estimates derived from different models explored as well as the probable interpretation of such estimates.

The results found in this assessment are then discussed in Chapter 4 in relation to the uncertainties involved and other studies that have been conducted. This chapter also details the possible public health impact of the assessment in terms of policy and presents reactions of policy makers to the results and method.

Chapter 2

METHODS

This chapter discusses the different steps in the methodology of risk assessment used in this study. It starts with the description of the study area and its characteristics followed by the definition of the study design, the variables and its sources and the manner by which the data were collected. Data quality and management, and quality assurance of the instruments of air pollution monitoring are likewise considered. Finally, the health risk assessment model and the method of risk assessment are discussed in detail. A brief review of literature introduces each component of the model followed by the description of procedures used for the purpose of this study.

Data utilized in the study are secondary data derived from various local and international sources. Local information was culled primarily from documents of the Department of Health (DOH), the Department of Environment and Natural Resources (DENR), the National Statistics Office (NSO), and studies conducted by local researchers. To obtain a clearer picture of the 1995 demographic, health and environment scenario, the data from the abovementioned sources were compared against the other for similarity and consistency.

2.1. Study Area and Population Characteristics

The study is carried out for the whole of Metropolitan Manila, Philippines, also known as the National Capital Region. Metropolitan Manila is composed of 17 cities and municipalities. It is situated in the southern part of the biggest island of the country that is the Luzon Island. Originally comprising only the City of Manila, due to rapid urbanization and industrialization of its surroundings in the past 30 years, the metropolis now include 8 cities and 9 municipalities. Among the 17 cities and municipalities, the largest in terms of population size is Quezon City (1,989,419

persons) that accounts for about one-fifth of the total population of Metropolitan Manila.

The population was estimated at about 9,454,040 as of 1995 with population density of 14,928 people per square kilometer. This was an increase of about 1.5 million from 1990 with a 12,550 population density. In the year 2000, the population has increased to approximately 10 million with an estimated 15,617 people per square kilometre (*Wallerstein, 1999*). The average annual growth rate for the period 1990-1995 was recorded at 3.30%. It is projected that if this rate continues, the population is expected to double within 21 years. However, estimate of the growth rate in the past 5 years was only about 2.02%. The population is a young population, the median age of which is 22.9 years in 1995. The age-sex pyramid for the metropolis shows a relatively higher proportion of population aged 15 to 29 years old (65%) that may be due to a large number of in-migrants in these age groups. The number of households increased slightly from 1.5 million in 1990 to approximately 1.98 million in 1995. The average household size in 1995 was 4.7 persons, lower than in 1990 at 5 persons. The simple literacy rate (basic reading and writing skills) for ten years old and over is quite high at 98.8%. Functional literacy rate (includes computational skills) is a bit lower at 92.4% as of 1994. The majority of the population of 15 years and over who worked at anytime during the past year was engaged in the service industry followed by manufacturing (*NSO Census, 1995*).

2.2. Study Design and Definition of Variables Used

This study is a risk assessment exercise wherein estimations of mortality impacts due to particulate air pollution have been computed among different population groups. Particulate matter, 10μm^3, was used as an indicator of the air pollution situation in Metropolitan Manila. As regards to indicators for impact of the air pollution scenario, mortality, morbidity, life expectancy and life years gained were utilized. Mortality and morbidity was used as measures of divergence from health in the affected population while life expectancy and life years gained was used as gauge for health gain. Mortality, as opposed to morbidity, refers to deaths in the affected population while the latter refers to illness. This is obtained by dividing the number of deaths that occurred in a particular time and place by the population at

that same time and place multiplied by a factor of 1000. Although mortality as an indicator reflects status of health services in an area, it indicated severest effect of the factor being studied. Life expectancy maybe defined as the average lifespan of an individual from birth (website reference life expectancy html). It reflected the average liveable years a person may reach given the combination of the environmental, social, and technological setting at his or her year of birth. Life years gained is an indicator that reflected the years added from the baseline life expectancies. The gain in life expectancy is said to be a good measure when comparing with other hazards like the lifestyle risk factors. The life years gained measure, on the other hand, is dependent on the size of the population (*Hurley et. al., 2000*).

The following table summarizes the variables and data that were used and their definitions in the context of this project:

***Table 2.1**: Summary of the Data Used and Level of Aggregation*

Population Characteristic	Definition	Level of Aggregation	Source	Time Reference
Age	Based on dates of births, intervals of age is constructed	Census Tract – metropolitan and city level	Population Census	1995
Sex	Male/Female	Census Tract	Population Census	1995
Socio-economic Status	Based on educational attainment of the household head	Census Tract – metropolitan and city level	Population Census	1995
ENVIRONMENT CHARACTERISTICS				
Particulate Matter,10μm (PM_{10})	Suspended particulate with aerodynamic diameter of 10μm	Metropolitan-wide annual averages	Environmental Management Bureau	1995
MORTALITY DATA				
Age	Total and age-specific numbers of deaths/midyear population per 1000	Metropolitan and city level	Vital registration system	1995

Table 2.1: Summary of the Data Used and Level of Aggregation (Continued)

Population Characteristic	Definition	Level of Aggregation	Source	Time Reference
Cause of Death by age group	Number of deaths per cause/midyear population for each age group per 1000. Specifically for cardio-pulmonary diseases,	Metropolitan level	Vital registration system	1995

The mortality data from the death registration system were used as the basis for computation of the health effects attributable to air pollution using a life table approach for the analysis (rationale for its use is explained section 2.6.4.1). The mortality outcome variable is expressed in terms of years of life lost or gained due to improvement in the air pollution situation and loss in years of life expectancy. Educational attainment is used as socio-economic status indicator.

This study included all 17 cities and municipalities for the Metropolitan Manila-wide analysis. However, in the individual analysis of the cities and municipalities, only 9 cities and municipalities were included due to the limited coverage of air quality monitoring network.

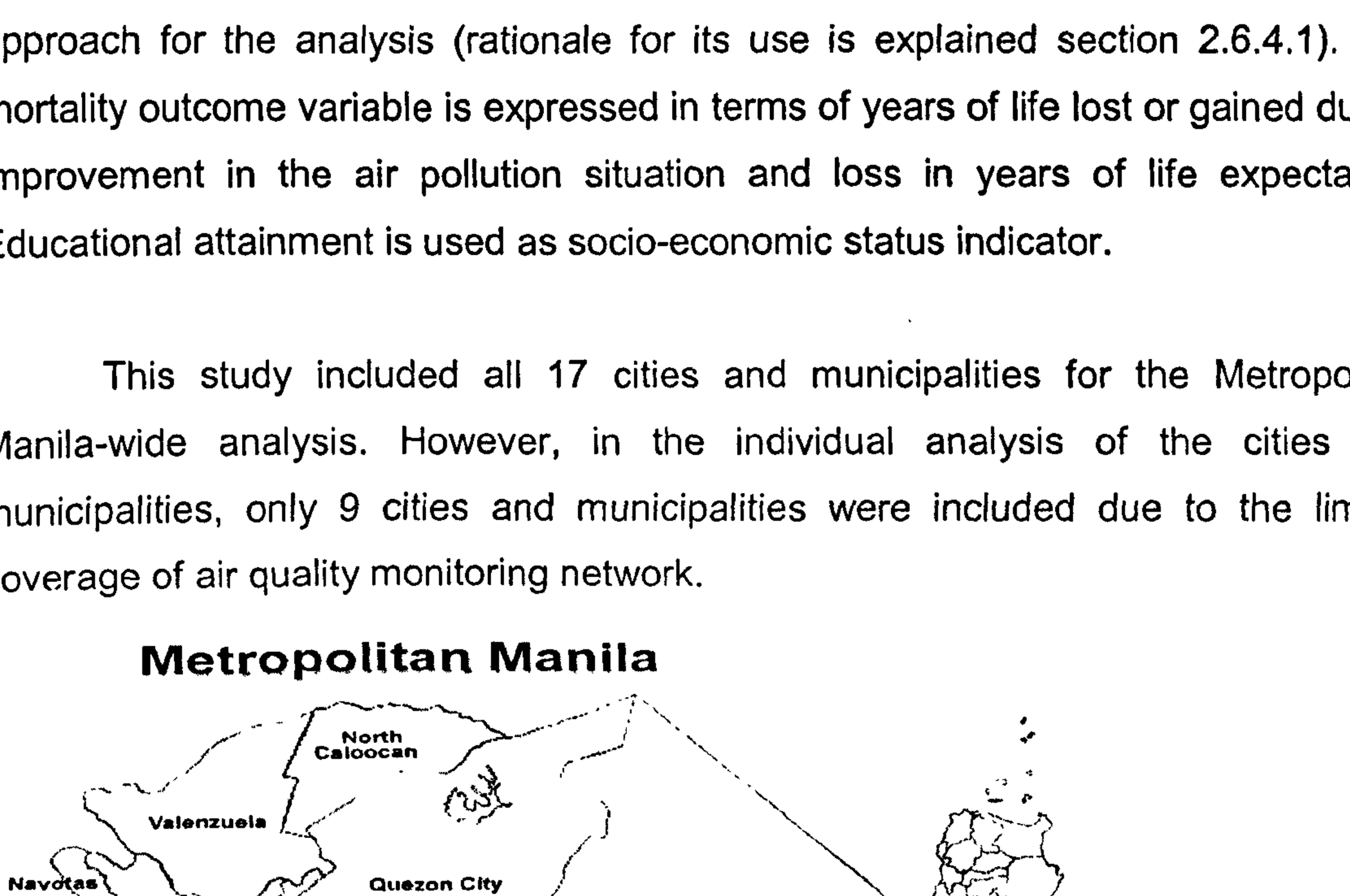


Figure no. 2.1: *Location of Metropolitan Manila in the Philippines*

2.3. Data Collection and Quality: Demographic, Mortality, and Exposure Data

Population data of the whole of Metropolitan Manila are available from the Population Censuses conducted in 1990 and 1995 by the National Census and Statistics office. This national census covered 11.4 million households in 1990 and 13.5 million in 1995 for the whole Philippines. Mortality data for 1995 are also available in the same office as part of the vital registration system of the country. A study of death registration in the Philippines showed that half of infant deaths are not routinely registered especially in the more distant rural areas. In fact in some regions overall death registration is as low as 20-40 %. However, the same study claimed that in the National Capital Region, this under-registration is almost negligible (*WB, 1996; Flieger. et. al., 1994*). This alleged completeness in death registration in Metro Manila is attributed to the vigilant enforcement of the death registration system in hospitals, health centres and clinics considering that the central office of the National Statistics Office which handles the registration system is located in Metro Manila.

The process of death registration starts from the office of the Civil Registrar of the city/municipality. All deaths are required to be registered within 30 days. It is the responsibility of the physician who last attended to the deceased or the administrator of the hospital or clinic where the person died to prepare the death certificate and to certify the cause of death. The death certificate is forwarded within 48 hours after death to the health officer who examines it and affixes his signature. The health officer orders its registration in the office of Civil Registrar which, in turn, sends the information to the National Statistics Centre (*Lourdes Hufana, 2000*).

The exposure data in the ambient air for Metropolitan Manila were taken from the Environmental Management Bureau (EMB). The EMB is the policy and regulatory arm of the Department of Environment and Natural Resources (DENR). The DENR-NCR office is the agency in-charge of the operations of the monitoring stations and all results are submitted to the EMB. Although, there were twelve monitoring stations in the whole of the metropolis at that time in 1995, particulate matter 10μm (PM_{10}) was only collected in two of these monitoring stations. The other ten stations collected total suspended particulates instead. Apart from particulates,

all the monitoring stations also collected ambient levels of sulphur dioxide (SO_2), nitrogen oxide (NO), nitrogen dioxide (NO_2), ozone (O_3), carbon monoxide (CO) and lead (Pb). However, the collection of all levels from all these other pollutants was not as regular as it was with the particulates. Also, according to the DENR personnel, only eight of the twelve stations including the two stations which collect the PM_{10}, were operational most of the time. The other four stations had equipment and maintenance problems all the time, thus they were not considered by the DENR to provide accurate and consistent results. Monthly results from the eight stations were given to the EMB which, in turn, calculated the annual averages for each station. However, clarification of this calculation and the way by which missing values were dealt with was lacking and could not be furnished.

2.4. Quality Control for the Air Pollution Measurements

According to the DENR, to ensure that the results of the monitoring stations were of high quality, several quality control procedures were undertaken. The reference method for the determination of suspended particulates in the ambient air was the gravimetric high volume method using high volume sampling equipment. The equipment from the eight functional monitoring stations was inspected regularly and followed a strict preventive maintenance programme. Quality assurance also involved regular periodic calibration, duplicate checks and split sampling including a reliable recording keeping system. In addition, on-site system survey, independent performance audits, inter-laboratory comparison and periodic evaluation of the internal quality assurance data were undertaken. The frequency of sampling for particulates was 24 hours per day every six days done throughout the year. The data was processed in the DENR-NCR office before transmission to the EMB which provided the information used in this study.

The sampling intakes were considered to be at street level and the measurement points were at about 2.5 meters from the street for all sites. Their location was based on the following criteria (*EMB-DENR, 1994*):

1. The site must be located in a populated area;
2. Several sites must be located downwind of the major emission sources;

3. The sampler intake must be exposed to the pollutants in an open area.

2.5. Data Management

For this study, the data management including data imports, checks for consistency and preliminary preparation of files is handled using EXCEL software. All data were provided by the sources mentioned in table form and could easily be manipulated in EXCEL. EXCEL has also been used in other risk assessment projects on air pollution (*Cifuentes et. al., 2001*). Exposure levels for nine of the 17 cities and municipalities were interpolated by the geographical information system using ARCVIEW and ARCINFO.

2.6. Building the Environmental Health Risk Assessment Model for Particulate Air Pollution

In this project, the USEPA model of risk assessment provides a sound basis for model building that is extended to include more complexity. This part of the thesis reviews each component of the risk assessment equation in terms of air pollution and provides a description of the methods by which the data were used and analyzed in each component. Although, enough description of the exposure response and exposure assessment components are contained in this section, the details of these components including a thorough review of the literature are presented in Chapters 3 and 4.

2.6.1. Assessing the Exposure-Response Relationship per Unit Increase in Air Pollution

In this section, the principles of the method by which the estimates were calculated and chosen are described. The explanation and its implementation are set out in Chapter 3. In the course of this description, some studies regarding the relationship between air pollution and mortality are cited.

The exposure response function is essential for calculating the attributable number of cases/deaths in health risk assessment. Here it is derived directly from epidemiological studies (not needing extrapolation from animal toxicological studies).

The assessment of these studies to arrive at the estimates is, therefore, extremely important. The World Health Organization Guideline on the evaluation and use of epidemiological studies in health risk assessment has recommended that all epidemiological information must be systematically reviewed in order to attain the appropriate and most reliable exposure-response relationship. (*WHO, 2000*)

In assessing the exposure-response relationship in this project, two types of study designs are considered, the time series and the cohort studies. To estimate long-term effects of particulate air pollution on mortality, the cohort studies were given more weight the reasons for which are discussed in Chapter 3. Thus, the main exposure-response functions used are based on these cohort studies. As mentioned in the literature review, three cohort studies, all of which were done in the United States could provide the estimates needed.

Considering the effects in different age groups, evidence from the time series studies has been found. Studies from the Netherlands, Spain and the United States have found higher relative risks for daily mortality for populations in the older age group. For the elderly, the relative risks were found to be 1.09 and 1.26 for Cincinnati, Ohio and Amsterdam respectively. While the relative risk found for total daily mortality in these two studies was 1.06 per 100 $\mu g/m^3$ increase in PM_{10}. In Spain, the relative risks found for total daily mortality were 1.009 per 10 $\mu g/m^3$ increase in black smoke and for the elderly, 1.008. Higher risks were seen for certain cause-specific mortality such as cardiovascular and respiratory mortality especially pneumonia. (*Schwartz, 1994; Verhoeff et. al., 1996; Ballester et. al., 1996*)

For children, Woodruff et al had reported a relative risk of 1.04 per 10 $\mu g/m^3$ change in PM_{10} for infant mortality and Bobak et al reported relative risk of 1.95 per 50 $\mu g/m^3$ change in TSP for post neonatal respiratory mortality. (*Woodruff et. al., 1997; Bobak et. al., 1999*)

Most studies on air pollution control for the effect of socio-economic status and age either through the design of the study or the analysis. Very few studies

have tried to investigate the potential differences in mortality experience among different socio-economic classes as affected by air pollution. Two recent studies, one by Gouveia and the other by O'Neill, are time series studies investigating effects of particulate pollution on mortality as modified by socio-economic classes. Gouveia in a time series study has found a difference in relative risks per 10 $\mu g/m^3$ PM_{10} for all cause mortality in the elderly as affected by air pollution between the poorer areas (~1.02) as compared to the rich ones (~1.05) (*Gouveia, 1997*). O'Neill's findings showed that stronger associations between PM_{10} and daily mortality were seen among the low socio-economic classes than the middle and high socio-economic classes (*O'Neill, 2000*).

For this study, a thorough review of the literature and a pooled estimate of these studies have been performed to arrive at definitive exposure response coefficients. The review started with the cohort studies, the coefficients of which form the basis of the exposure response estimates ultimately used. The three main cohort studies were compared according to several aspects. The same was done to the two main infants' studies. These aspects include the sample size, design and frame, sources and methods of obtaining information regarding the outcome measures, response and refusal rates, description of the sources of the exposure measures and control of the potential confounder variables. After such comparisons, a pooled estimate was undertaken. It is a way of quantitatively assessing the strength of association across studies by combining the measures of effect (*Schwartz, 1994*). To derive a summary estimate, a weighted average of the log relative risks in the two adult cohort studies that were considered and an estimated standard error were calculated. Reasons for the exclusion of the study by Abbey et al from the pooled estimates are given in Chapter 3 section 3.2. An inverse variance weighting is utilized. From the variance of the summary estimate, confidence intervals, z-statistics and the p value were calculated (*Lecture Notes, 2001*). These calculations were done using STATA statistical software. All the values used in this assessment were converted to the relative risk per 10 $\mu g/m^3$ PM_{10}. Conversions are detailed in Chapter 3 section 1. For the two infant studies, no meta-analysis was undertaken because the estimates in the two studies were very similar. Thus the pooled

estimate based on the meta-analysis of the two adult cohort studies and the estimate from the Woodruff study which was more extensive, were used as exposure-response coefficients for this risk assessment.

With regards the time series studies, the pooled estimates of several studies from the developed countries including the air pollution-mortality study of 90 cities in the United States were reviewed as well. Because of its extensive coverage, the estimate from the study of 90 cities in the USA (*Samet et. al., 2000*) was utilized to compare with the estimates from the developing countries. This forms part of the evaluation of the validity of extrapolating from developed countries to developing countries. To assess the differential effects of age and with evidence from time series studies on the effects on the elderly, a meta-analysis similar to that described earlier, was undertaken. The purpose for this meta-analysis was to come-up with a pooled estimate for the effect of PM^{10} on mortality among the elderly.

In terms of the time series studies from developing countries, a systematic review was made. For this review, guidelines for review of time series studies as prepared by Shwartz and by Thurston and Kinney were followed. Comparison of the studies with regards to several aspects including autocorrelation and overdispersion was undertaken (*Schwartz, 1994; Thurston et. al., 1995*). Following this review, pooled estimates for all ages and the elderly were calculated from the meta-analyses of the qualifying studies. For the purpose of this assessment, differences in effect estimating age, only studies which presented both all age and elderly mortality were used. The ratio of the relative risks for the effects on the all age and elderly mortality was then applied to the pooled estimate from the adult cohort studies. A summary of the results of these pooled estimates including the estimates used in this assessment can be seen towards the end of Chapter 3.

2.6.2. Selecting the Exposure Targets

The WHO Air Quality Guidelines have been used as targets for air quality improvements and as baseline levels for estimating air pollution attributable health impact (*Ostro, 1994*). By subtracting the air quality guideline from the measured

pollutant level in the area that is considered the excess, an air pollution improvement scenario is created. In turn, this excess level is multiplied by the dose-response coefficient and the exposed population to get the risk estimate. Several studies including those done in Bangkok, Manila and Jakarta have made used of such a method.

In this study, three pollution reduction scenarios were utilized to show different degrees of benefits. The three pollution reduction scenarios make use of the WHO air quality guideline of 50 $\mu g/m^3$ PM_{10}, the National Standard of 60 $\mu g/m^3$ PM_{10} (EMB-DENR, 2000) and finally a reduction to background level. The annual averages from each of the eight monitoring stations were averaged to come-up with an average level for the whole of Metropolitan Manila. Since there were only two stations that monitor PM_{10}, the TSP levels from the other six stations were converted to PM_{10}. This annual average level for 1995 was used as the basis of the exposure assessment.

In addition, since there were eight monitoring stations all over the metropolis, an attempt to map out the levels of PM_{10} in each city was made with the use of the Geographical Information System (GIS). Proximal interpolation through the use of the triangulation interpolation method was employed to estimate the exposure level of each city or municipality. However, only 12 of the cities and municipalities could be included in this interpolation as the others were not within the pathways of the triangles formed by the stations. This technique is described in Chapter 4.

2.6.3. Estimating the Exposed Population

By using the data from a single monitoring station or as summarized from several stations, the exposure is assumed to be non-differential for the whole population. However, in reality, people's exposure to ambient air pollution levels is largely dependent on the types of activities and on the behaviour of individuals in that population.

The exposed population used for this study was the general population from which rates attributable to air pollution were ascertained. The adult portion of the

general population was then categorized according to socio-economic class as defined by educational level. Differential effects among these socio-economic classes were also investigated. The percentages of the population in Metropolitan Manila with regards to the different educational levels were defined from the census. However, since the death registration system did not have information on socio-economic classes, the study adapted estimates on mortality rate ratios among different educational groups from the United States and was applied to the Metropolitan Manila population. The details and discussion of this issue can be read in Chapter 3.

2.6.4. The Modified Risk Assessment Model

Krzyzanowski proposed a method for assessing the extent of exposure and effects of air pollution. This framework for assessment of impact was mainly based on the Covello-Merkhofer model with four stages: assessment of release of pollutant; assessment of exposure; assessment of the consequence; and the risk estimation. He emphasized that the use of epidemiological data is critical in assessing the consequences of exposure to the pollutants. The model used in this study utilized only epidemiological data. (*Covello et. al., 1993; Krzyzanowski, 1997*)

In order to account for some heterogeneity in the exposed population, certain variables were considered in the model. It is the aim of this study to look into variation of effects by age and socio-economic status.

The basic principle of risk estimation can be illustrated by the following equation:

Attributable Number of Cases = Exposure-response coefficient X excess exposure level X exposed population X baseline mortality rates

The exposure-response coefficients were deduced from several epidemiological studies, including some meta analysis done for this purpose. This part of the equation was explained earlier. It is important to note at this point the use

of exposure-response coefficients from acute effects studies particularly the daily time series in risk assessment. It should be pointed out that due to the 'harvesting' assumption of such studies and problems with estimating directly the loss of person-time, since this data are not available in such studies, associated with the estimated extra deaths, dose-response coefficients from daily time series studies must be used with caution. It is not recommended for use in quantifying long-term health impacts (*McMicheal et. al., 1998*). Further discussion of this point could be seen in Chapter 3. Thus in this study, exposure-response coefficients from chronic effects studies were given emphasis. Although, coefficients of the association between mortality and air pollution are found for PM_{10} and SO_2 in the two cohort studies, the exposure-response function will be limited to PM_{10} only as a general indicator of air pollution since the data of the other pollutants in Metro Manila were not regularly collected. Therefore all studies that made use of other forms of particulate matter such as total suspended particulates (TSP) and black smoke (BS) were converted to PM_{10}. This study also standardised changes in the response variable per $10\mu g/m^3$ of PM_{10}.

As discussed earlier, the excess exposure level was based on the WHO Air Quality Guidelines, the National air quality standard drawn by the Department of Environmental and Natural Resources (DENR) and the background level of PM_{10}. The air quality monitoring data for 1995 were used as exposure levels and the exposed population were the Metropolitan Manila population.

However, it is in the definition of the exposed population and to a lesser extent, the dose-response coefficients where some refinements were made. Since some evidence on the effect of age exists, risk estimation regarding certain age groups was made, particularly for elderly and infant mortality. A caveat must be attached, though, in using studies from developed countries, as developing countries' exposure scenarios are not exactly similar. The reanalysis of the two main adult cohort studies provided information regarding the effect of socio-economic status on mortality associated with particulate pollution. These exposure-response coefficients were used for this study.

2.6.4.1 The Life Table Approach

The standard life table approach has been applied extensively in estimating life expectancy from age-specific death rates (*Newell et. al., 1999)*. Brunekreef, in 1997, has found usefulness of this method in predicting the life expectancy of Dutch Males with particulate pollution reduction in comparison with no reduction (*Brunekreef, 1997*). Nevalainen et al likewise utilised this approach in a similar study in Norway, which included analysis of competing causes of death. (*Nevalainen et. al., 1998*) Application of this approach enabled the assessment of the effects on mortality of different reductions of particulate pollution. Furthermore, it compared these effects in terms of life years gained, life shortening and deaths avoided, measures that can be used to do economic valuation in the future (*Hurley et. al., 2000*). Given the prevailing mortality conditions, which in this study are based on the 1995 mortality rates, life expectancy is the number of years expected to be lived by those born in a particular year (*Newell et. al., 1999*). It is from the life expectancy table that the three other measures mentioned earlier were derived.

Construction of a life table involves only the age specific death rates as raw data. Probabilities of dying and surviving are calculated from this raw data. Other columns in the table can subsequently be computed. Two types of life tables are usually derived, namely a complete life table based on single-year age groups and an abridged life table wherein most of the ages are grouped, (e.g. 5-year age group) (*Ibid.*). In this study, except for the calculation of life expectancy if the effects were delayed, the abridged life table method was generally used. The life tables were constructed in the EXCEL software programme. The following are the steps as summarized from Newell, examples of the tables can be seen in the Annexes (*Ibid.*).

Age Specific Death Rate

Probability of survival. The inverse value of the probability of dying, derived by deducting the latter from the constant value 1

No. of deaths in the cohort. Derived by multiplying the probability of dying to the cohort population

Cumulative number of Years Lived Derived by multiplying the number of years lived with the cohort pop

Age Grp	Population	Cases	Rate	N	nQx	nPx	lx	ndx	Lx	Tx	Ex
(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Under 1	129201	2548	0 0197	1	0.019567	0.980433	100000	1956.686	98825 99	7177581	71.77581
1 to 4	455632	877	0.0019	4	0 007676	0 992324	98043.31	752.5371	390367.2	7078755	72.20029
5 to 9	478433	308	0 0006	5	0 003214	0 996786	97290.78	312.6604	485672.2	6688388	68.74638
10 to 14	433907	261	0.0006	5	0.003003	0.996997	96978.12	291.2293	484162 5	6202716	63.95996
15 to 19	556091	304	0.0005	5	0.00273	0 99727	96686.89	263.9199	482774.6	5718554	59.14508
20 to 24	581667	430	0.0007	5	0.003689	0 996311	96422.97	355.7481	481225.5	5235779	54.30012
25 to 29	523779	510	0.0010	5	0.004857	0.995143	96067.22	466.5642	479169.7	4754553	49.49194
30 to 34	412827	620	0.0015	5	0.007481	0.992519	95600.66	715.199	476215.3	4275384	44.72128
35 to 39	340462	644	0.0019	5	0.009413	0 990587	94885.46	893.178	472194.3	3799168	40.03952

Interval. Number of years interval between upper and lower age limit

Probability of dying. Derived utilizing age-specific death rates, age group interval, and population of particular age group

Index or Cohort population. A radix value, usually 100000 is assigned the first age group and the succeeding values are derived by multiplying the radix by the probability of survival

Number of Years Lived by population in that particular age group. Derived by multiplying probability of survival with the cohort population

Life Expectancy of Particular Age group. Derived by dividing the cumulative number of years lived with the index of cohort population

1. 1995 age and sex specific death rates and midyear populations were obtained from the death registration system, columns 2-4. The crude age specific death rate is seen in column 4.
2. N is the number of years in each category or the age grouping, column 5.
3. The (q_x) probability of dying and the (p_x) probability of surviving are then calculated. Since the number of population is needed for this calculation and only the midyear populations are available, adjustments are made for both the numerators and the denominators. The assumption is for each death to have contributed half of the year to the interval N, hence nQ_x and nP_x
4. The lx is the index or cohort population. A radix, in this case -100,000, is chosen at the beginning and then multiplied with nPx or the probability of survival to the end of the interval.
5. The d_x is the number of deaths per age group in the cohort population. This number of deaths is calculated by multiplying the lx with the nP_x.
6. The L_x is the number of years lived by the population in that age group.
7. The T_x is the cumulative number of years lived for each age group.
8. Finally the E_x is the life expectancy at a particular age. This calculated by dividing the T_x for each group by the lx for the same age group.
9. The same method is undertaken when dealing with single age grouping.

See sample of LIFE TABLE in Annex 1

2.6.4.2. Incorporating the Exposure-Response Coefficients and Exposure Levels

The basic methodology in this study is described as follows: From the life table, the attributable fraction of mortality due to PM_{10} for each age group was calculated by multiplying each age specific mortality rate with the respective exposure-response coefficient for the age group. For example, a relative risk 1.04 was used for infants (*Woodruff et. al., 1997*), 1.05 for 30-64 year olds (*Pope et. al., 1997; Dockery et. al., 1993*) and 1.053 for 65 years old and over. The exposure-response coefficient was dependent on the pollution reduction scenario, i.e. for 50 $\mu g/m^3$ PM_{10}: The 1995 annual average exposure level was 95.6 $\mu g/m^3$ PM_{10}. The excess PM_{10} was 45.6 $\mu g/m^3$. The exposure-response coefficient of 1.04/10 $\mu g/m^3$ PM_{10} for the infants is applied to a difference in PM_{10} of 45.6 $\mu g/m^3$. Thus the total relative risk due to PM_{10} for the infants was 1.19. These conversions are more fully explained in Chapter 3 section 1. This procedure was done for 30 years olds and above as well. For those who were between 2 to 29 years old, no reduction was made. The rates attributable to air pollution (assuming causality) were then subtracted within each age group before completion of the life table. Hence, a life expectancy based on a certain reduction scenario was produced. This alternative life expectancy was compared with the existing (1995) life expectancy. This comparison yielded the gains in life expectancy when PM_{10} pollution was reduced. A sample of the life table involving this process is seen in Annex 2.

Example: From the 1995 Mortality data

Male/Age Group: under 1 year old Population: 140200 Number of deaths: 3634 Crude Death Rate (M_x): 0.0259 N (numbers of years in the category): 1 Relative Risk to be used: 1.19 M_x – clean (death rate in reduced Pollution by 45.6 $\mu g/m^3$ PM_{10}): (0.0259) – (0.0259 x (1.19-1/1.19)) = 0.0217 This M_x-clean is used as the new death rate for life table analysis for the under 1 age group.	Calculation of the Relative Risk to be used: 1. From the Woodruff study: Relative risk of 1.04 for 10 $\mu g/m^3$ Increase in PM_{10} 2. Excess PM_{10} to be considered: 45.6 $\mu g/m^3$ 3. Conversion: (log 1.04/10 $\mu g/m^3$) x 45.6 $\mu g/m^3$ =0.1788, when exponentiated it is equal to 1.19

Mortality data were also available for twelve of the 17 cities and municipalities within Metropolitan Manila. Differential exposure levels were likewise deduced from the GIS for these cities and municipalities. Using the basic

method described above, life expectancies in different pollution scenarios were calculated for each of these cities and municipalities.

2.6.4.3. Socio-Economic Status Variable as Measured by Educational Attainment

As expressed earlier, this assessment considered the effects of PM_{10} pollution on the mortality of different socio-economic groups. In the reanalysis of the US adult cohort studies, it was found that there is effect modification on the effect of PM_{10} on mortality by educational level. The educational levels were divided to low (less than high school education, < 8 years schooling), middle (high school, 8-12 years schooling) and high (more than high school, >12 years schooling). The relative risks found were 1.35,1.23 and 1.06 respectively comparing the most polluted with the least polluted cities, a difference of 24.5 µg/m3 PM_{10}. (*Krewski et. al., 2000*)

The census data in Metropolitan Manila contained the educational level categories similar to the classification mentioned above. Unfortunately, the death registration system did not have such information. Thus baseline mortality rates by socio-economic status cannot be calculated. Hence, in this assessment, in order to estimate public health impact as measured by the mortality among different socio-economic groups in Metropolitan Manila, mortality rates ratios by socio-economic status from the US needed to be estimated. This was done by assuming that the relative risks by educational levels would be the same as that found in the literature, in this case from the US. These US mortality rates ratios together with the educational level census data of Metropolitan Manila were applied to the Metropolitan Manila mortality data to generate baseline rates.

A significant outcome of the reanalysis of the Six-Cities and American Cancer Society studies is the demonstration of differences in mortality risks of particulate pollution of persons in relation to different educational categories as indicated earlier in. Thus, revealing some vulnerability among those who are less educated. In computing the life expectancies for different socio-economic classes in Metropolitan Manila based on educational attainment in this study, two assumptions had to be made. Although the percentages of people in the three different educational classes were available for the whole Metropolitan Manila

and for the nine cities within the metropolis from the census data, the mortality data classified according to educational attainment were not, since this is not a feature of the Philippine death certificate. No direct evidence from the Metropolitan Manila data regarding differences in mortality among socio-economic groups exists. Hence in this regard, mortality rate ratios from another study and population would have to be adapted in order to illustrate the variability of impact of effects of particulate pollution in different sectors of the Metro Manila population. A most appropriate study for this purpose is the national longitudinal study of mortality in the United States (*Sorlie et. al., 1995*). This study was chosen firstly, because the categories between the Metropolitan Manila educational data were similar and interaction between educational classes and air pollution had been shown. Secondly, the mortality differences seen between educational classes were much bigger in the US than in the other developed countries as will be seen later. Studies of measures of socio-economic status in developing countries have been shown to have greater mortality differences among these socio-economic groups than in developed countries (*Duncan et. Al, 1995.; Mackenbach et. al., 1999; Shkolnikov et. al., 1998*). The Philippines, being a developing country, would most probably be in a similar category.

This US study of over 500,000 people shows interaction of age with educational attainment. It demonstrates that at different age groups, the effect of educational attainment on mortality is different as well. Another US study has investigated educational level and mortality based on a smaller sample of 3617 individuals, however age interaction with education was not explored (*Lantz et. al., 1998*). For the larger US longitudinal study, the rate ratios were higher for men than for the women. Because of its bigger sample size, a more precise estimate and a demonstration of age interactions with education which may be more realistic, these estimates in the US longitudinal study are used in this study.

The mortality rates among different educational groups in Metropolitan Manila estimated by transposition of the US mortality rate ratios must be interpreted with caution and recognized as a limitation of this study for a couple of reasons. First of all, there are socio-cultural differences between the two countries that might affect the mortality rates in different educational levels. The magnitude of the differences in the estimates because of these socio-cultural

differences, however, could not be entirely predicted. Secondly, the proportion of the population in the respective educational categories is different in the US study with that of the Metropolitan Manila population. For the males, the proportions of middle and high educational groups in the Metropolitan Manila population were 49% and 23% respectively. In the US study, the proportions were 54% for the middle and 25% for the high educational groups. The middle and high educational groups among males had higher proportions in the US than in the Philippines by approximately 5% and 2% respectively. With the low educational level, a higher proportion was found in the Metropolitan Manila (28%) than the US (21%). For the females, the differences were slightly greater (*NSO Census, 1997; Sorlie et. al., 1995; Shkolnikov et. al., 1998*). Hence, the mortality rates among educational groups in Metropolitan Manila based on the US mortality rate ratios could have modestly underestimated the real differences.

Since the US rate ratios had eight classifications of educational attainment, the rate ratios to make up three classifications instead were approximated by calculating the average of the rate ratios for each educational level. Then, using the highest educational level (16 years +) as the basis, estimated rate ratios between this base (high) and the low and middle socio-economic classes were computed. The following are the rate ratios used for this estimation:

Table 2.2: *U.S. Adult Mortality Rate Ratios according to educational attainment by Sex and age group, 1979-1989.*

AGE – Educational Level		MALE	FEMALE
25 – 44 y/o	Low	2.67	2.09
	Middle	1.79	1.38
	High	1.00	1.00
45 – 64 y/o	Low	1.90	1.66
	Middle	1.47	1.20
	High	1.00	1.00
65 + y/o	Low	1.34	1.21
	Middle	1.19	1.09
	High	1.00	1.00

These different rate ratios were then applied to the Metropolitan Manila adult population mortality rates, ages 25 years old and above, accordingly to compute for the life expectancy at 25 years old. In computing for the mortality rates per educational group, apart from the US mortality rate ratios, the

percentage of the population in each educational group was likewise considered. As can be seen in Table 3.15, the difference in the life expectancy at 25 years old between the high and middle educational levels is bigger than the difference in life expectancy between middle and low educational levels for both males and females. A slightly wider difference between the different educational levels among males than females is also observed.

For comparison, mortality rate ratios from another country were likewise applied to the Metropolitan Manila adult population mortality rates. A Swedish study of more than 32,800 men and women, age 25-64 years, found mortality rate ratios of 1.29 (1.2-1.48) for low educational category as compared to the high and 1.21 (1.03-1.42) for the middle educational category as compared to the high among males (*Sundquist et. al., 1997*). Age interaction was not explored in this study. When applied to the Metropolitan Manila adult population mortality rates, the differences in life expectancies between age 25-64 years old are less than the US counterpart estimates. These differences are less because the rate ratios found in the Swedish data is also much less than the US.

With the estimation of the mortality rates of the three educational categories in Metropolitan Manila, the relative risks from the reanalysis of the US adult cohort studies were applied in the same manner as described in section 2.6.4.2. Again, different pollution scenarios were assessed and compared. In addition, a no effect modification scenario was also explored. Instead of using the different relative risks from the reanalysis study, the overall relative risk, 1.05/10 $\mu g/m^3$ PM_{10} increase, was adapted. In this scenario, this overall relative risk was applied in all three educational categories.

2.6.4.4. Cardio-Pulmonary Causes of Death

Deaths in this category could be responsible for much of the excess risk as it is the most biologically plausible. For this reason, the exposure-response relationship of this category of deaths has been studied and described. In this study, the impact of PM_{10} on such causes of death among the Metropolitan Manila population is calculated. Life expectancy calculations were made if only the cardio-pulmonary causes of death (ICD 460-519 for respiratory causes, ICD 401-459 for cardiovascular causes) were modified instead of the total deaths.

The ACS and Six-Cities pooled estimate for cardio-pulmonary causes of death and the Woodruff estimate for respiratory causes of death were used for this purpose. Age-specific death rates due to cardio-pulmonary causes were separated out from the deaths due to other causes (except in the case of the infant deaths, only deaths due to respiratory causes were considered). Then, these cardio-pulmonary age specific death rates were modified much the same way as the basic method of calculating for the attributable fraction described earlier. The resulting modified cardio-pulmonary age specific death rates were added to the age specific death rates from other causes. These new age specific death rates were then used to calculate life expectancies much the way as described in section 2.6.4.2.

2.6.4.5. Population Living 1995 and the 1995 Birth Cohort

Other types of measures, aside from life expectancy, such as percent of people expected to survive to a particular age, reduction in premature deaths and life years gained are considered in this section. These effect measures could help policy makers in better understanding the magnitude of the problem as will become apparent in the discussion chapter. In addition, comparison of scenarios of delayed effects is explored, similar to that done in the Institute of Occupational Medicine Report on England and Wales (*Hurley, 2000*). Two types of populations are studied. The first is the birth cohort who was born in 1995 followed throughout their lives and the other is the population alive in 1995 followed for the next five years in order to investigate the benefits in the short term. In both cases, it is assumed that the age-specific mortality rates seen in 1995 are constant for the years of follow-up as well as the birth and migration rates.

The same basic method as outlined in section 2.6.4.1 was employed in the calculations of life expectancies in these scenarios from which the other effects measures mentioned earlier were derived. Delayed effects and the unabridged life table were utilized. For the population alive in 1995, effects in the short term were measured, by calculating the gains in life expectancy, life years and reduction in expected number of deaths within five years of implementation of the intervention. Both immediate and delayed effects scenarios were explored and an unabridged life table was utilized.

In summary, the mortality data had been collected from the death registration system with the annual average for 1995 of particulates provided by the DENR-EMB that operated the eight monitoring stations. The quality of both data sets is considered adequate for use in this study. The components of health risk assessment were outlined and discussed according to the way they were utilized. The life table approach was deemed to be an appropriate technique. The basic method of adjusting for the attributable fraction of deaths was discussed in the context of the life table approach. Finally, different ways by which the basic method was modified were detailed, i.e. age effects, socio-economic effects, additive model etc.

Chapter 3

RESULTS

This chapter begins by discussing specific demographic characteristics of Metro Manila residents in 1995 relevant to the indicators being utilized. This is followed by a presentation on the conversions made in order to standardise all the exposure-response coefficients and the summary of these coefficients. The literature of these coefficients was reviewed in section 1.3.1. The issue of the transferability of coefficients is discussed, exposure assessment data is presented and finally, the main findings of these results are discussed.

3.1 Exposure-Response Coefficients

3.1.1. Description of Metro Manila Population

In 1995, population density in the National Capital Region, mainly composed of the Metro Manila areas involved in the study, grew from 202.3 persons per sq. km. to 228.7 per sq. km. This increase is likely attributable to in migration and natural population growth which increased from 2.98 % in the decade of the 80's to 3.30 % from 1990 to 1995. For 1995, there were 94.2 males for every 100 females. Majority of the population belonged to the 20 to 24 years age group, followed closely by the 15 to 19 years age group then by the 25 to 29 year olds. There are more children below 15 years of age than there are adults 30 to 54 year olds. An estimated 1.7 million families were residing in NCR Metro Manila in 1994 earning an average annual income of 173,599.00 pesos. That is roughly an income of 14,400.00 pesos per month per family. The annual per capita poverty threshold for 1994 was 11,230.00 pesos. At this value, 141,671 families composed of approximately 1.9 million individuals, were considered to be below the poverty threshold. Simple literacy for 10 years old and over was at 98.8% in 1994 while functional literacy for 10 years old to 64 years old was at 92.4%. As introduced in

Chapter 1, road vehicles have greatly increased from 600,000 to almost a million from 1990-1994 with diesel fuelled vehicles increasing by more than 40%. Daily volume of vehicles exceeds 140,000 to 150,000 and in the busiest roads, 11,000 to 12,000 vehicles per hour are accommodated. Metro Manila is continuously influenced by sea and land breeze circulation owing to the 2 large bodies of water located in the western side, Manila Bay, and south-eastern side, Laguna de Bay, bordering the area.

3.1.2 Conversions

Different studies on particulate air pollution used different metrics of measurements for particulate matter. Some, like the American Cancer Society study by Pope et al (*Pope et. al., 1995*) and the Time Series study in Mexico City study used $PM_{2.5}$ as the particulate size (*Borja-Aburto et. al., 1998*) while others, as the New Delhi study, used TSP or total suspended particulates (*Cropper et. al., 1997*). Naturally this depends on the available monitoring data in the respective study areas. Apart from particle size, investigators also opted, to present results by ranges depending on how the data were analyzed. Some investigators present by change per 10 $\mu g/m^3$ or per 100 $\mu g/m^3$ TSP or per 10 $\mu g/m^3$ PM_{10} or $PM_{2.5}$. Thus before applying these exposure-response functions to the study at hand, there is a need to standardize these functions. In this case, all exposure-response functions will be converted to a per 10 $\mu g/m^3$ PM_{10} mainly because the Metropolitan Manila data is in PM_{10} or is converted to such from TSP. For the conversion of total suspended particulates (TSP) or black smoke (BS) to PM_{10}, the relationship 0.55TSP or BS = PM_{10} was used (*Torres et. al., 1996*) whereas the conversion of $PM_{2.5}$ to PM_{10} used the relationship 0.70PM10 = $PM_{2.5}$ (*Fang et. al., 1999*).

The relative risk shows how an increase in particulate air pollution is expected to increase the mortality based on the results of the analysis. For example, a relative risk of 1.26 for mortality as in the six-city cohort study (*Dockery et. al., 1993*), for a difference of 18.6 $\mu g/m^3$ PM_{10} between the least and most polluted cities, means that the mortality rate is increased by 26%. For a time Series study as in the Sao Paolo study with a relative risk of 1.005 per 10 $\mu g/m^3$, this means that when the daily

concentration of PM_{10} increase by 10 $\mu g/m^3$, the daily mortality increases by 0.5% in this case, with a 2 day lag (*Gouveia, 1997*). In the latter example, the coefficient is estimated from a Poisson regression model:

$$\text{Relative risk} = e^{\beta x}$$

where:

e = the exponential function

β = the estimated parameter for the variable x from the Poisson regression

x = the increment of variable x for which the relative risk is being calculated.

This underlying relationship must be borne in mind when converting the reported relative risk to a relative risk from a different exposure difference (*Chestnut et. al., 1998*). For example, the Santiago study reported that the $\beta = 0.075$ for x measured in 100 $\mu g/m^3$ units. In order to estimate the relative risk per 10 $\mu g/m^3$, the β coefficient must be divided by 100, and then multiplied by 10 and subsequently exponentiated.

$$\text{Relative risk} = e^{(0.075/100) \times 10}$$

$$= e^{0.0075}$$

$$= 1.0075$$

Where β is not presented, it can readily be calculated by this equation:

β = log relative risk / exposure increment.

Thus in the case where the relative risk is given as in the six-cities studies, to convert the increment to 10 g/m3 PM10, the log of the relative risk must be taken and then divided by the reported difference between the least and most polluted cities and multiplied by 10. Example:

$$\text{Relative risk} = e^{(\beta \times 18.6)}$$

$$= 1.26$$

Change in RR for 10 $\mu g/m^3$ = (log 1.26/18.6) x 10

= 0.12425 when exponentiated = 1.13

After all the exposure-response coefficients and their standard errors or relative risks and their confidence intervals were converted to relative risks or coefficients per 10 $\mu g/m^3$, they were utilized for calculating pooled estimates used in this study.

3.1.3 The Exposure Response Functions

The following summarizes the exposure-response functions used for this study and shows the calculations for the differential estimates with regards to age effect. The method by which the elderly estimate was arrived at is to simply approximate the differences in both the developing countries' and developed countries' pooled estimates as will be shown. The US cohort reanalysis reported no modification of the relative risk by age. Pope, nevertheless, argued that since the relative risk found in the cohort studies was small; this implied that the long term cumulative effects were most likely to be observed in the older age groups with relatively higher baseline mortality risks (*Pope, 2000*). In addition, several time series studies do indicate such an effect, so the potential of the magnitude of effect of introducing an age interaction was explored.

The final estimates for the infants and adults used in this study are based primarily on the American cohort studies. However, in case of the relative risk for the elderly as seen above, this was based on the differences seen between total population estimates and elderly estimates from the time series studies in both developed and developing cities. The difference in the estimates between the total and elderly populations from the developing countries was given more weight in calculating the estimate for the elderly used in this study than the difference of estimates in the developed countries. This arbitrary weighting was done to reflect similarity with the estimates from the developing countries.

The calculations for coming up with the exposure response functions for the 30–64 years old and 65 and over separately are done in the following manner. The relative risk for the 30-64 years old is based on both relative risk for the 65 years and above and the number of deaths in the two age categories.

Air Pollution and Mortality Time series studies:

RRt = relative risk for 30 and above

RRy = relative risk for 30-64 yrs old

RRo = relative risk for 65 and above

Developing countries:

RRo = 1.010

RRt = 1.008

Ratio between the two RR: 1.00198

Developed countries:

RRo = 1.011

RRt = 1.009

Ratio between the two RR: 1.00198

Developing countries ratio is given more weight than the developed countries (70% vs. 30% - arbitrary). However, since the ratios between the total and elderly relative risks in the two groups of studies are the same, this weighting would not make any difference. Therefore the ratio to be used to adjust the relative risk from the cohort studies to reflect the exposure response function for the elderly is 1.00198.

RRc – relative risk from cohort studies: 1.05

D – total deaths in Metropolitan Manila: 35,100

d- excess deaths

c – concentration

Dy – deaths of 30-64 yrs old in Metropolitan Manila: 18,423

Do – deaths of 65 and above in Metropolitan Manila: 16,677

@ - ratio from the Time Series Studies: 1.00198

RRo = RRc*@ = 1.05*1.00198 = 1.052

The relative risk for the young was calculated as the value that would yield the appropriate weighted average for the total population.

RRy-1/RRy = ((D*(RRc-1/RRc)) – (Do*(RRo-1/RRo)))/ Dy

RRy-1/RRy = ((35,100*(1.05-1/1.05)) – (16,677*(1.052-1/1.052)))/18,423

RRy-1/RRy = 847.09/18,423

RRy-1/RRy = 0.046

RRy = 1/(1-0.046)

RRy = 1.048

Therefore, the estimates are: Relative risk for the young: 1.048

Relative risk for the old : 1.052

***Table 3.1**: Summary of Exposure-Response Functions*

Type of Studies	Risk Ratios with confidence intervals per 10 $\mu g/m^3$ PM_{10} (*Excess Risk)
A. From Cohort studies in the USA	
1. Dockery et al : 6-cities study	1.09(1.03-1.16)
2. Pope et al : ACS study	1.045 (1.025 – 1.068)
3. Woodruff et al: infant mortality	1.04 (1.02 – 1.07)
4. POOLED ESTIMATE: adults only	1.05 (1.03 – 1.07)
B. From Time Series Studies in Developed Countries	
POOLED ESTIMATE from 3 studies: Holland, Spain & Cincinnati, Ohio, USA – all ages	1.009 (1.005-1.012)
2. POOLED ESTIMATE from 3 studies: Finland, Spain & Cincinnati, Ohio, USA - =/> 65 years old	1.011(1.005-1.016)
C. From Time Series Studies in Developing Countries	
1. POOLED ESTIMATE from 3 studies: Bangkok, Mexico City and Santiago – all ages	1.008(1.006-1.010)
2. POOLED ESTIMATE from 4 studies: Bangkok, Mexico City, and Santiago – 65 years old and over	1.010(1.007-1.012).
D. *ESTIMATES TO BE USED IN THIS STUDY*	
1. Infants (=/< 1 year old)	1.04 (1.02 – 1.07)
2. Adult (=/> 30 yrs, < 65 yrs)	1.048 (1.028 – 1.068)
3. Elderly (=/> 65 yrs)	1.052 (1.032 – 1.072)
4. Adult (=/>30 yrs)	1.05 (1.03-1.07)

3.1.4 Transferability of Coefficients:

An important issue in risk assessment is the appropriateness of adapting coefficients from other situations to the population concerned in the original studies. The transferability of the exposure-response coefficients is even made more complicated by the fact that the coefficients being used are from a developed country and utilized for estimation in a developing country. In the Philippines, studies linking particulate pollution and mortality are not available. Thus, for the purpose of this study, exposure-response coefficients from the US cohort studies are applied. However, as discussed earlier, there are possible reasons for these coefficients not to be transferred from the US population to Metropolitan Manila. These reasons are dealt with accordingly in the following paragraphs.

First of all, the quality of exposure in the two areas may be different. The second major issue involves several other differences that are found between developed and developing countries. These differences include the different level of exposure to pollutants, the different level of mortality and the different general level of the socio-economic conditions between these two countries. Each of these aspects is discussed.

Although pollution mix and range of sources might be different, generally, the main sources of particulate air pollution, especially the fine particles, could be classified as stationary and mobile. A third type which is considered a minor source are called area sources or smaller stationary sources such as domestic burning and unpaved roads. Stationary or point sources refer principally to industries, factories and power plants while mobile sources refer to those which are mostly traffic-related i.e. cars, trucks etc. These stationary and mobile sources are considered the most common for both developed and developing countries. Certainly, this is true for the cities of Metropolitan Manila (*WB, 1996*) and the cities in Western and Eastern United States (*US EPA, 1997*) where the cohort studies were conducted.

With regards the components of particulate pollution mix, in the comparison of the western and eastern cities of the United States in the ACS study, it was reported that the mixture of pollutants, i.e. sulphates in the $PM_{2.5}$ fraction is much

greater in the east than in the west (*Hurley et. al., 2000*). In spite of such differences, the effects seen in the cohort studies in terms of risk per unit of PM_{10} were comparable in magnitude (*Pope et. al., 1995*). Moreover, Harrison and Yin's review of airborne particulate matter studies showed that generally, similar components, as measured by elemental proportions, were present in the samples taken from cities in developed countries. They pointed out, however, that in some less developed countries, it might be different, for example with regards to wind-blown coarse dust being present. In the case of the Philippines, the latter could be a factor because of unpaved roads. But in the same review, the authors expressed that there is no evidence that any single component of the particulate matter is responsible for the apparent adverse health effects seen. Hence, the pollution mixture does not seem to have any bearing with regards the effects (*Harrison et. al., 2000*). In addition, evidence from the time series studies conducted in less developed cities, show that the mix may not be totally relevant in terms of the magnitude of effects. This latter aspect will be dealt with next.

The second issue is rooted in the differences between developed and developing countries. There is no direct evidence from the cohort studies, ACS and Six-Cities studies, to judge the applicability of the coefficients from the developed to developing countries. However, such evidence exists in other types of studies, namely the time series studies. Time series studies have been conducted in both developed and developing countries as seen from the review earlier. These time series studies have found that the magnitude of effects on mortality due to particulates is quite close in both cities of developing and developed countries (20-NMAPPS and Pooled estimate).

As can be observed from the coefficients derived from the time series studies from both cities in the developed and developing countries, the magnitude of effect is only slightly different. The NMAPPS study revealed a 0.5% increase with 10 $\mu g/m^3$ PM_{10} (*Samet et. al., 2000*) while the pooled estimate from the time series studies of four cities in developing countries showed a 0.8% increase with 10 $\mu g/m^3$. The developing countries' point estimate is slightly higher than the developed countries.

However the estimate from the developing countries lies within the range of the confidence interval of the NMAPPS study, the 20-city study's CI, 95% interval, 0.05, 0.92, thus this difference is not significant. And so for a cautious, conservative assumption, the coefficients are treated the same. The pooled estimate from the developing countries had not overestimated the effect. Therefore, although the range of exposures, the levels of mortality, and the general level of socio-economic conditions are different between the two countries, the effect and its magnitude remain comparable. With such evidence and by analogy with the cohort studies, the effects between the two countries are plausibly similar. This is supporting evidence for using the Six-Cities and ACS coefficients for estimating public health impact in Metropolitan Manila.

3.2 Exposure Assessment

The magnitude of the impact of any environmental health hazard depends on the levels and range of exposure for which the assessment is required to estimate the attributable cases (*WHO, 2000*). Consequently, the calculation of the health effects of air pollution only has meaning in respect to a change in the exposure level of the air quality being considered. Ostro specified three ways by which the change in air quality could be defined. The first way is by considering the change from the present level to some ambient air quality standard, i.e. WHO guideline, and local standards. The second change is in terms of specifying a specific percent or amount reduction as was done in the UK assessment with $10\mu g/m^3$ of PM_{10} reduction from present levels. The third way involves an assumption regarding the relationship of air quality changes with changes in emissions. In this latter, for example, reducing the total emissions of a particular pollutant by 10 percent can be estimated to result in a 10 percent reduction in ambient air levels, given a simple linear relationship between the two. (*Torres et. al., 1996*)

With regard to the exposure levels of the population, two methods were utilized to assign exposure levels to the population or sub-population. With an existing ambient air monitoring system, levels of exposure could be assigned to the population concerned. An average of the levels from the all the monitoring stations

may be used for the whole population's exposure level, as was done in this study. If groups of people in a specific area are being examined, the levels from the nearest local monitoring station were designated for that particular area and population. The other method is employed as an alternative to monitors when these latter are non-existent or unreliable. Dispersion models can be developed when emissions and its sources can be characterized and meteorological and topographic characteristics are combined. From such dispersion models, levels of exposure were deduced and assigned to the population/individuals depending on the location of residence (*Ostro, 1996*). These two concerns, the change in exposure level considered and the level of exposure assigned to the Metropolitan Manila population are dealt with in the following paragraphs.

In this study, particulate matter, specifically PM_{10} was chosen as an indicator of air pollution. PM_{10} has also been used extensively in other air pollution risk assessments. (*Brunekreef, 1997; Hurley, et. al., 2000; Kunzli, et. al., 2001*). In several time series studies, PM_{10} had been shown to be robust in its relationship with mortality even when other pollutants were included in the model and also independent of other pollutants (*Schwartz, 1994; Verhoeff et. al., 1996; Ponka et. al., 1998*). However, it must be pointed out as well that using PM_{10} alone would probably underestimate the total effects of air pollution. Nevertheless, its use would give an indication of the magnitude of effects.

Since a fairly reliable ambient air quality monitoring system existed at the year chosen for the study, annual average levels from the stations were used for the exposure levels of the Metropolitan Manila population. There were eight (8) monitoring stations functioning for the whole of Metropolitan Manila during the year chosen for this study, 1995. All of the stations were considered background monitors. Only two of the eight stations monitored for PM_{10}, the rest were for TSP. The two PM_{10} stations were primarily set up for the thermal power station located in the area. There were twelve monitoring stations originally set-up all through out the metropolis, however due to lack of funds and poor maintenance, the four stations

were eventually closed down. Thus none of the data from those four monitoring stations was considered here.

Table 3.2: *Selected results from selected stations*

Station	Location	1995 Annual Mean Level (ug/m^3) *TSP, **PM_{10}	1995 Minimum-Maximum Levels (ug/m^3) for the year
1. Valenzuela	Northwest	296 *	131 – 454
2. PAGASA QC	Northeast	133 *	43 – 310
3. EDSA/DPWH QC	Northeast	234*	64 – 401
4. Ermita, Manila	West	174*	77 – 302
5. Viejo, Makati	Central	210*	41 – 485
6. Municipal Hall, LP	South	118*	68 – 198
7. Severina Cpd	South	59**	158***
8. Sucat	South	64**	231***

***maximum daily mean

The above table lists the stations and their location, the 1995 Annual Mean Level and the Minimum - Maximum Levels of Total Suspended Particulates and Particulate Matter, 10um:

The stations with total suspended particulates are converted to particulate matter, 10μm by the relationship 0.55TSP = PM_{10} to standardise calculations with the exposure-response functions (WB, 1996; Ostro, 1996). The following are the results of the conversions:

Table 3.3: Levels Converted to PM_{10}

Station	PM_{10} in ug/m^3 (minimum-maximum &/or *maximum daily mean
1. Valenzuela	163 (72-250)
2. PAGASA QC	73 (24-171)
3.EDSA/DPWH QC	129 (35-221)
4.Ermita Manila	96 (42-166)
5.Viejo, Makati	116 (23-267)
6.Municipal Hall, LP	65 (37-109)
7.Severina Cpd	59* (158*)
8.Sucat	64* (231*)

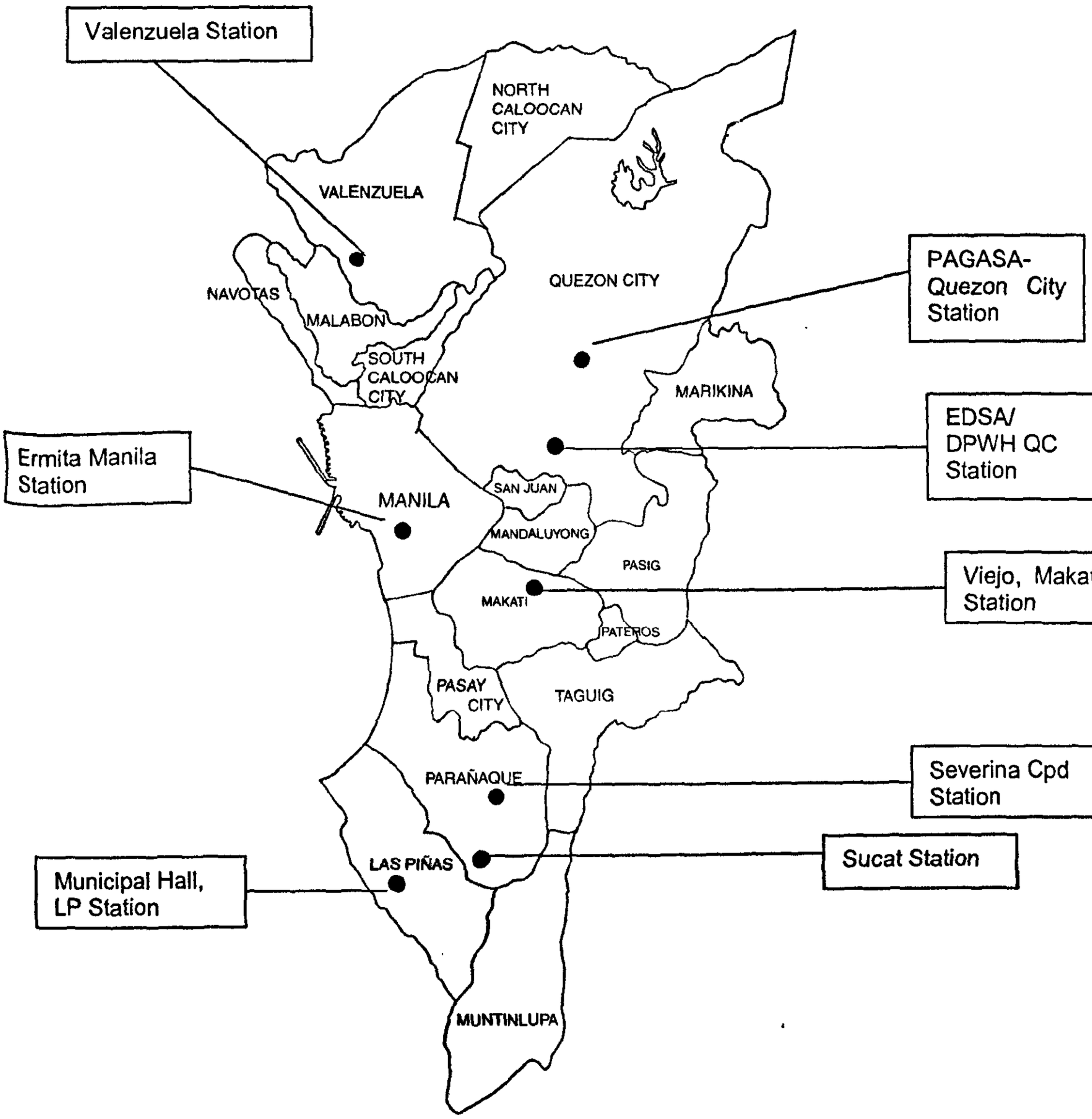


***Figure no. 3.1**: Air Monitoring Stations in Metropolitan Manila, 1995*

From these levels, an average exposure level for PM_{10} for the whole of Metropolitan Manila was computed. The average exposure level is 95.6 $\mu g/m^3$ PM_{10}. From the minimum and maximum levels of the six stations with these data, average minimum and maximum levels are also deduced. The average minimum and maximum levels of exposure levels were 38.8 – 197.3 $\mu g/m^3$ PM_{10}.

Apart from assessing the exposure level for the whole of Metropolitan Manila, an attempt to describe the exposure of specific cities within the metropolis was done. The differences in ambient particulate levels in the monitoring stations in the cities in the north with the cities in the south of the metropolis would probably result as well to differences in attributable cases. As mentioned earlier, the simplest method by which exposure could be assigned to an individual or population was to take the ambient level of pollutants at the nearest local monitoring station. This method is common in air pollution studies, i.e. time series studies, cross-sectional studies. However, for the purpose of this study, a more complex method, i.e. spatial interpolation technique was utilized in order to arrive at levels of exposure in each individual city. Spatial interpolation is the prediction of levels at unmeasured locations from measured data for the surrounding locations. Spatial interpolation can be done by either making use of discrete techniques which utilize boundaries or methods which consider continuous spatial change described by mathematical modelling. The more common methods of spatial interpolation are proximal interpolation, spline interpolation and kriging. Proximal interpolation is an example of the discrete technique while spline interpolation and kriging are examples of the latter. Both spline interpolation and kriging require numerous data and data points within the areas of concern and are more complicated techniques. (*Burrough, 1991; Elliot et. al., 1992*) Thus, the method used in this study was proximal interpolation primarily because of the type of data available i.e. annual averages from each monitoring station, the number of data points, only 8 data points, and its distribution.

In this method, proximal interpolation was employed with the help of the Geographic Information System. This interpolation technique is based on the assumption of a disjunctive variation. Each area or region is said to have an

internally uniform distribution of the pollutant and be separated from the next area or region by a narrow zone of abrupt change. (*Ibid.*) The concept involved the calculation of the best estimate of exposure for points in the area that did not have the direct measurement. The best estimates for those points in the area were based on all the known data points nearest to it. The Thiessen polygons divided the area up depending on the configuration of these existing data points. Specifically, a triangulation interpolation network was applied. From three known data points, levels in the areas within those three points were interpolated. The area weighted average exposure was calculated for each city from the series of interpolated data areas within. Average city level exposures were made since the health data to which it would be related to were also at that level.

Although, the validity of this technique is limited e.g. no account is taken of local or random variation in the data, it is the best that could be done with the data available. (*Burrough, 1991*) There were only eight data points in the whole metropolitan area and some cities were not within the triangles formed by the eight data points. Hence, only 12 of the 17 cities and municipalities had estimated measurements. This is illustrated in Figure 3.2.

The following are the mean exposure levels of the cities and municipalities where the triangulation technique can be applied. Only 12 of the 17 cities and municipalities are included because the areas of interpolation by triangulation did not cover the other 5 cities and municipalities.

Table 3.4: Cities and Municipalities within Metropolitan Manila with individual Annual PM_{10} Levels, 1995.

City/Municipality	PM_{10} Mean Exposure Level(ug/m^3)
1. Valenzuela	153
2. Malabon	146
3. Caloocan City	137
4. Quezon City	124
5. Manila City	109
6. San Juan	117
7. Mandaluyong	113
8. Makati City	99
9. Pasay City	81
10. Parañaque	71
11. Taguig	87
12. Las Piñas	66

In order to compare the impact of different levels of particulate air pollution on mortality, 'excess PM_{10}' was calculated depending on the desired exposure or pollution reduction scenario. These were the changes in air quality considered. Three ways of calculating excess PM_{10} were employed in this project. The first method is based on the international guideline of 50 $\mu g/m^3$ PM_{10}. This guideline is subtracted from the levels obtained in the preceding table to come up with the excess PM_{10}. A second method is similar to the first one, however using instead the Philippine National Annual Guideline. The National Annual Mean Guideline of PM_{10} in the Philippines is 60 ug/m^3. The third way is to first subtract a background level of TSP (in this case, based on the Canadian background level – 30 $\mu g/m^3$ TSP) (*WB, 1996*) and then multiplying the product with the 0.55 to get the PM_{10} level. Thus the third scenario is a reduction to background levels. Excess exposure levels from the three methods are applied for this risk assessment. The use of the different exposure levels in calculating the excess PM_{10} could assist in standard-setting of particulate air pollution in Metropolitan Manila and at the same time, test the sensitivity of the assumptions of the assessment.

Table 3.5: *Excess PM_{10} for each city/municipality: Pollution Reduction Scenario 1 (reduction to 50 ug/m^3 PM_{10})*

Cities/ Municipalities	Excess PM_{10} ($\mu g/m^3$)
1.Valenzuela	103
2. Malabon	96
3. Caloocan City	87
4. Quezon City	74
5. Manila City	59
6. San Juan	67
7. Mandaluyong	63
8. Makati City	49
9. Pasay City	31
10. Parañaque	21
11. Taguig	37
12. Las Piñas	16
Metropolitan Manila	45.6 $\mu g/m^3$ PM_{10}

Table 3.6: Excess PM_{10} of Each City: Pollution Reduction Scenario 2 (reduction to 60 $\mu g/m^3$)

Cities/Municipalities	Excess PM_{10} ($\mu g/m^3$)
1. Valenzuela	93
2. Malabon	86
3. Caloocan City	77
4. Quezon City	64
5. Manila City	49
6. San Juan	57
7. Mandaluyong	53
8. Makati City	39
9. Pasay City	21
10. Parañaque	11
11. Taguig	27
12. Las Piñas	6
Metropolitan Manila	35.6 $\mu g/m^3$

Table 3.7: Excess PM_{10} per city/municipality: Pollution Reduction 3 (reduction to background level)

Cities/Municipalities	Excess PM_{10} ($\mu g/m^3$)
1. Valenzuela	136
2. Malabon	129
3. Caloocan City	121
4. Quezon City	108
5. Manila City	92
6. San Juan	100
7. Mandaluyong	97
8. Makati City	83
9. Pasay City	65
10. Parañaque	54
11. Taguig	70
12. Las Piñas	50
Metropolitan Manila	66.35

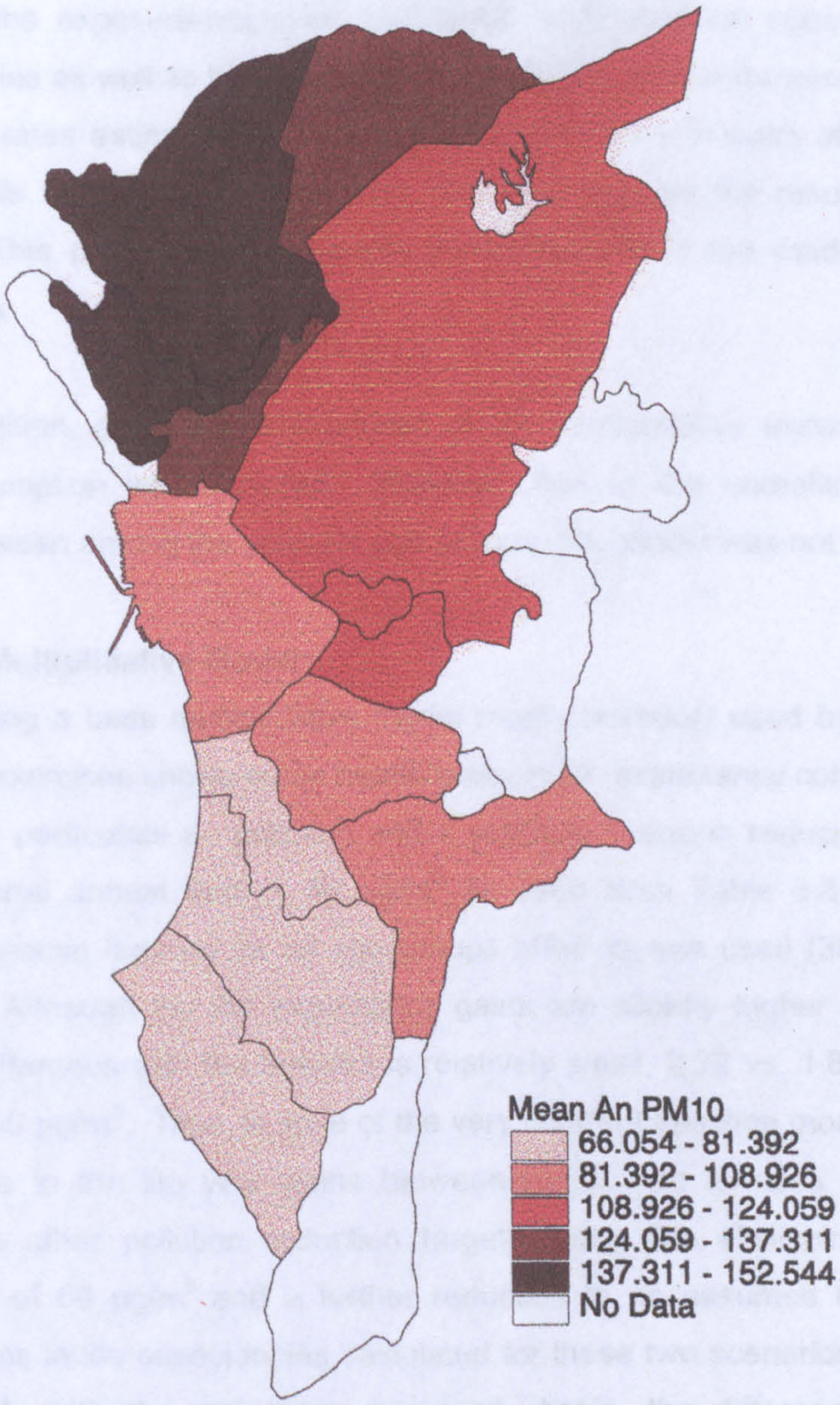


***Figure no. 3.2**: Estimated Annual Average Levels of PM_{10} in Some Cities of Metropolitan Manila, 1995*

3.3. Main Findings in the Application of the Exposure-Response Coefficients and Exposure Levels to the Mortality data

After the exposure-response coefficients were decided upon based on previous studies as well as the exposure levels, these coefficients were utilized to arrive at the rates estimated under cleaner air conditions in each affected age group, and life tables were constructed. The following are the results of such application. This presentation of results starts from the base model to more complex ones.

In addition, given the arbitrariness of the multiplicative assumption, the additive assumption was explored. However, due to the unrealistically high relative risks seen among the younger age groups, this model was not pursued.

3.3.1. Base Multiplicative Model

Adapting a base multiplicative model most commonly used by other risk assessment exercises shows some health gains in life expectancy comparing the 1995 level of particulate air pollution and a pollution scenario reducing PM_{10} to the international annual limit of 50 $\mu g/m^3$ as seen from Table 3.8. Only one exposure-response function for all age groups affected was used (30 years old and above). Although the life expectancy gains are slightly higher among the males, the difference with the females is relatively small, 2.22 vs. 1.88 years for reduction to 50 $\mu g/m^3$. Thus, in spite of the very different baseline mortality rates, the difference in the life year gains between males and females is minimal. Applying two other pollution reduction targets, using the Philippine National Annual Limit of 60 $\mu g/m^3$ and a further reduction to an assumed background level, the gains in life expectancies calculated for these two scenarios increased proportionately with the reductions proposed. Again, the difference between males and females is relatively small. Decreasing the particulate levels to background level yielded more than 2.5 years difference with the no reduction scenario for both males and females. Clearly, the differences in life year gains are sensitive to the levels of proposed reduction. These observations are seen in Table 3.9.

***Table 3.8**: Comparison of Two Pollution Scenarios using only 1 relative risk coefficient – 1.05 – for 30 years and above for Metropolitan Manila, 1995 (in life expectancy, years)*

Pollution Reduction Scenario	MALE	Difference	FEMALE	Difference
1. No Reduction	64.10		71.77	
2. Reduction to 50 $\mu g/m^3$	66.32 (65.4-67.15)	2.22	73.65 (72.90-74.32)	1.88

***Table 3.9**: Comparison of Different Pollution Scenarios, PM_{10}, using only 1 relative risk coefficient 1.05 – for 30 years and above, for Metropolitan Manila, 1995 (in life expectancy, years)*

Pollution Reduction Scenario	MALE	Difference	FEMALE	Difference
1. No Reduction	64.10		71.77	
2. Reduction to 60 $\mu g/m^3$	65.83 (65.13-66.48)	1.73	73.25 (72.67-73.78)	1.48
3. Reduction to Background Level (16.5 $\mu g/m^3$)	67.29 (66.08-68.52)	3.19	74.44 (73.46-75.41)	2.67

3.3.2. Heterogeneity: Age Effects

Exploring some heterogeneity with regards age, different exposure-response functions were applied to age groups known to be affected by air pollution. As mentioned earlier, this assumption is based on evidence from some time series studies, a cohort study of infants as well as biological mechanism studies of vulnerable groups, which are not necessarily older groups but those with pre-existing disease conditions in both animal and human studies. Relative risk coefficients for infants and those 65 years old and over were slightly different from the coefficients used for 30-64 years olds. The method by which this was calculated is detailed in Chapter 2.

Table 3.10 shows that more than two years are gained in life expectancy for both male and female with this method in the pollution reduction to 50 µg/m3 scenario. Comparing the values with Table 3.8, the differences in life expectancy between the two models are 0.40 years for males and 0.29 years for females in favour of this model. These figures translate to about 4.80 months for males and 3.48 months for females. These differences may have been contributed largely by the contribution of the infant mortality.

According to the latest reanalysis of the Six-Cities and American Cancer Society Studies, for 30 years old and above, no difference was seen among the age groups. (*Krewski et. al., 2000*) Thus, evidence of differential effects is seen among the older age groups only in acute conditions. Making use of these findings, only one relative risk coefficient was applied for those over 30 years old, a different coefficient for infants and no effect for other age groups. The results, seen in Table 3.12, show slightly higher figures for both males and females than those seen in Tables 3.8 and 3.9 but almost similar with those seen in Tables 3.10 and 3.11.

Investigating the sensitivity of the assumptions in this model, other pollution reduction levels were tested as well as seen in Table 3.11. As in the base multiplicative model, the differences in the health gains in life expectancies for both males and females increased or decreased proportionately with the amount of exposure reduction. Lessening the exposure to background levels revealed an increase of health gains in life expectancy of more than 3 years for both males and females. The base multiplicative model consistently shows more modest estimates of the effect of particulate air pollution than the models which attempt to account for some heterogeneity with regards to age.

In Tables 3.10 and 3.11, the relative risk used for 65 years and above was higher and a lower value was used for the 30-64 years old age group. The two relative risks had an average value of 1.05 for all the 30 years old and above. The increase in life expectancy in these tables was almost similar with the estimates found in Table 3.12 among the males. For the females, the estimates in Tables 3.10 and 3.11 were slightly higher than the estimates in Table 3.12. These results could probably be due to the differences in baseline mortality rates. The baseline mortality rates for the females were very much higher in the older age group than in the 30-64 years old age group. Thus, with the higher mortality rates and higher coefficient used for the older age group, a slight difference with the model using a single coefficient for 30 years and over is detected. Among the males, compared with the females, the baseline mortality rates for the 30-64 and older age groups were almost similar. Hence, in the model utilized for Tables 3.10 and 3.11, because of the lower coefficient used, the contribution of the 30-64 years old age group to the life expectancy calculation would be similar to the

contribution of the older age group where a higher coefficient was used. Thus comparing it with the estimates in Table 3.12 that made use of a single coefficient for 30 years old and above, similar gains in life expectancy was found. The contribution of the application of a coefficient in the infant age group is like wise apparent in the increase in life expectancy.

***Table 3.10**: Comparison of Two Pollution Scenarios using three different relative risk values (1.04 - infants, 1.048- 30-64 years old, 1.052- 65 years old and above) for corresponding age groups, for Metropolitan Manila, 1995 (in life expectancy, years)*

Pollution Reduction Scenario	MALE	Difference	FEMALE	Difference
1. No Reduction	64.10		71.77	
2. Reduction to 50 $\mu g/m^3$	66.72 (65.61-67.77)	2.62	73.96 (73.06-74.8)	2.19

***Table 3.11**: Comparison of Different Pollution Scenarios, PM_{10}, using three different relative risks values (1.04-infants, 1.048-30-64 years old, 1.052- 65 years old and above) for corresponding age groups, for Metropolitan Manila, 1995 (in life expectancy, years)*

Pollution Reduction Scenario	MALE	Difference	FEMALE	Difference
1.No Reduction	64.10		71.77	
2.Reduction to 60 $\mu g/m^3$	66.04 (65.22-66.80)	1.94	73.45 (72.77-74.06)	1.68
3.Reduction to Background level	67.67 (66.22-69.21)	3.57	74.79 (73.6-76.00)	3.02

***Table 3.12**: Comparison of Different Pollution Scenarios, PM_{10}, using two relative risk coefficients, 1.04/ 10 $\mu g/m^3$ for infants and 1.05/ 10 $\mu g/m^3$ for 30 yrs and above, for Metropolitan Manila, 1995 (in life expectancy, years)*

Pollution Reduction Scenario	MALE	Difference	FEMALE	Difference
1. No Reduction	64.1		71.77	
2. Reduction to 60 $\mu g/m^3$	66.05 (65.24-66.84)	1.95	73.44 (72.76-74.09)	1.67
3. Reduction to 50 $\mu g/m^3$	66.72 (65.61-67.76)	2.62	73.95 (73.04-74.77)	2.18
4. Reduction to Background level	67.69 (66.29-69.16)	3.59	74.78 (73.63-75.95)	3.01

Comparing the estimates of Metropolitan Manila adult life expectancies by education using the US mortality rate ratios with that of the life expectancies of different socio-economic classes by education in Finland and Norway, the inequality still remains higher in the Metropolitan Manila life expectancies using

the US estimates, more so for males than for females as can be seen in Table 3.17 (*Sihvonen et. al., 1998*). These Scandinavian countries are known to be more egalitarian than the United States. The bigger differences seen in using the US estimates may be more realistic due to the reasons stated earlier. These life expectancies among different levels of educational attainment are shown

***Table 3.13:** Life Expectancy Estimates at Age 25*

Socio-economic Class	Male	Difference with high socio-economic class	Female	Difference with high socio-economic class
Low	39.82	6.75	47.72	4.4
Middle	41.29	5.28	48.25	3.87
High	46.57		52.12	

Estimates are for different Socio-economic Classes according to Educational Attainment in Metropolitan Manila, Philippines using the US adult mortality rate ratio estimates, 1995.

***Table 3.14**: Life Expectancy Estimates at Age 25-64 yrs*

Sex - Socio-economic Class		US Estimates - difference		Swedish Estimates- difference	
Male	Low	30.93	2.25	31.71	0.69
	Middle	31.8	1.38	31.9	0.5
	High	33.18		32.4	
Female	Low	33.13	1.34	33.3	0.7
	Middle	33.7	0.77	33.6	0.4
	High	34.47		34	

Estimates are for different Socio-economic Classes according to Educational Attainment, Metropolitan Manila, Philippines using the US and Swedish adult mortality rate ratio estimates, 1995.

***Table 3.15:** Life Expectancy Estimates at Age 25-74 yrs*

Sex - Socio-economic Class		US Estimates - difference		Finland - difference		Norway - difference	
Male	Low	36.38	3.98	42	3.9	43.7	2.8
	Middle	37.61	2.75	44	1.9	45.1	1.4
	High	40.36		45.9		46.5	
Female	Low	40.62	2.42	46.6	1.2	46.9	1.1
	Middle	41.7	1.34	47.4	0.4	47.6	0.4
	High	43.04		47.8		48	

Estimates are for different Socio-economic Classes according to Educational Attainment, Metropolitan Manila, Philippines using the US adult mortality rate ratio estimates, 1995 and the life expectancies in Finland and Norway for the same age groups in the late 1980s.

Following the multiplicative model, calculations are made for life expectancies at age 25 years in different pollution reduction scenarios for the different educational categories. Two models were used for the purpose of comparison. The first model made used of the differential relative risks of mortality for particulate pollution and educational levels taken from the American Cancer Society (ACS) reanalysis study. The second model used the single ACS exposure-response coefficient. This coefficient was applied to the affected age

groups that were classified according to educational levels as explained earlier. The differences in life expectancies between decreased levels of particulate air pollution and the no reduction scenario are more similar among all educational categories using the single ACS exposure-response coefficient than in the model with the interaction for educational level. These estimates are shown in Tables 3.18 and 3.19. The health gains in life expectancy are also noticeably higher for the low and middle-educational groups than for the high-educational group. With the model using the interaction factors, gains of approximately two years or more are evident for the low and middle-educational groups in all three pollution reduction scenarios. In fact, in the reduction to background level, the gains could be as high as 6 years for the low educational groups and more than 4 years for the middle educational groups. While only one year or even less is seen among the high-educational group.

***Table 3.16**: Comparison of Pollution Reduction Scenarios (Using relative risks coefficients of 1.35, 1.23 and 1.06)*

Pollution Reduction Scenario	MALE			FEMALE		
	Low	Middle	High	Low	Middle	High
No Reduction	39.82	41.29	46.57	47.72	48.25	52.12
Reduction to 60 ug/m^3	43.26 (41.59-44.83)	43.5 (42.01-44.94)	47.13 (46.57-48.08)	50.61 (49.23-51.85)	50.03 (48.84-51.15)	52.57 (52.12-53.30)
Difference	3.44	2.21	0.56	2.89	1.78	0.45
Reduction to 50 ug/m^3	44.19 (42.15-46.17)	44.09 (42.21-45.93)	47.31 (46.57-48.53)	51.35 (49.70-52.87)	50.49 (49.00-51.90)	52.71 (52.12-53.67)
Difference	4.37	2.8	0.74	3.63	2.24	0.59
Reduction to back ground level	46.1 (43.18-48.73)	45.38 (42.7-47.97)	47.57 (46.57-49.39)	52.82 (50.54-54.75)	51.49 (49.38-53.44)	52.91 (52.12-54.32)
Difference	6.28	4.09	1.0	5.1	3.24	0.79

Comparisons are among Different Educational Levels Using relative risks coefficients of 1.35, 1.23 and 1.06 respectively for low, middle and high educational levels (in terms of life expectancy at age 25, years)

Table 3.17: *Comparison of Pollution Reduction Scenarios (Using the general relative risk coefficients of the ACS study)*

Pollution Reduction Scenario	MALE			FEMALE		
	Low	Middle	High	Low	Middle	High
No Reduction	39.82	41.29	46.57	47.72	48.25	52.12
Reduction to 60 μg/m^3	41.59 (40.8-42.41)	42.97 (42.21-43.76)	48.07 (47.40-48.76)	49.23 (48.56-49.92)	49.61 (49.00-50.23)	53.31 (52.77-53.84)
Difference	1.77	1.68	1.5	1.51	1.36	1.19
Reduction to 50 μg/m^3	42.15 (41.10-43.18)	43.5 (42.5-44.49)	48.54 (47.65-49.40)	49.7 (48.82-50.54)	50.03 (49.23-50.80)	53.69 (52.98-54.32)
Difference	2.33	2.21	1.97	1.98	1.78	1.57
Reduction to back ground level	43.18 (41.68-44.69)	44.49 (43.06-45.93)	49.49 (48.15-49.39)	50.54 (49.31-51.74)	50.80 (49.68-51.90)	54.32 (53.37-55.25)
Difference	3.36	3.2	2.82	2.82	2.55	2.2

Comparisons among Different Educational Levels Using the general relative risk coefficients of the ACS study: 1.17/35 μg/m^3 PM_{10} for 30 years and above, for low, middle and high educational levels (in terms of life expectancy, years).

3.3.3. Modifying the Cardio-Pulmonary Causes of Death

The ACS and the Six-Cities Studies also found significant relative risks attributable to particulate air pollution among cardio-pulmonary mortality. For the purpose of this thesis, a pooled estimate of the two studies was used which is 1.09 per 10 μg/m^3 PM_{10} for the 30 years and older mortality (*Pope et. al., 1997; Dockery et. al., 1993*). Woodruff et al, likewise, found a significant relative risk for respiratory causes of deaths among infants attributable to particulate air pollution. This relative risk of 1.2 per 10 μg/m^3 PM_{10} was also used here (*Woodruff et. al., 1997*).

The results of modifying the cardio-pulmonary causes of death are shown in Table 3.18. This table also compares the life expectancies modifying all cause-mortality rates. As expected, the differences between the life expectancies of 1995 exposure levels and those with lower particulate exposure levels modifying the all cause mortality are mostly bigger than those which modified only the cardio-pulmonary mortality except with the pollution reduction to 60 μg/m^3. The direction of the difference between the reduction in all cause mortality and the reduction in cardio-pulmonary causes of death depend on which age group dominates the baselines rates as well as the amount of reduction for each age group. The differences, however, are quite modest. This shows that the cardio-

pulmonary causes of deaths attributable to particulate air pollution dominate the attributable fraction in the all-cause mortality.

***Table 3.18**: Comparison of Pollution Reduction Scenarios modifying only the Cardio-Pulmonary Causes of Death and modifying All Cause Mortality*

I. Pollution Reduction Scenario: Modifying only the Cardio-Pulmonary Causes of Death				
	MALE	Differences	FEMALE	Differences
No Reduction	64.10		71.77	
Reduction to 60 $\mu g/m^3$	66.16	2.06	73.87	2.10
Reduction to 50 $\mu g/m^3$	66.68	2.58	73.47	1.70
Reduction to Background level	67.70	3.60	74.64	2.87
II. Pollution Reduction Scenario: Modifying All Causes of Death				
	MALE	Differences	FEMALE	Differences
No Reduction	64.1		71.77	
Reduction to 60 $\mu g/m^3$	66.05	1.95	73.44	1.67
Reduction to 50 $\mu g/m^3$	66.71	2.61	73.93	2.16
Reduction to Background level	67.69	3.59	74.77	3.00

3.3.4. 1995 Birth Cohort and Population Alive

A. 1995 Birth Cohort

Three pollution reduction scenarios for a single birth cohort born in 1995 followed throughout their lifetime are presented in Tables 3.19 and 3.20. In these scenarios, no differential age effects were applied to the adult groups (30 years and over), but the infants group had a different relative risk coefficient. As in Table 3.12, the life expectancy gains increase proportionately with increasing reduction of particulate pollution. For the males, expected survival to age 65 years increased by 3% to 5% comparing the no reduction scenario with the other three pollution reduction scenarios. A similar comparison in the expected survival to 75 years old category, over 5% to more than 9% increases were observed. More modest results are seen among the females.

***Table 3.19**: 1995 Birth Cohort, Males*

Pollution Reduction Scenario	Life Expectancy In years	Expected Survival to age 65 years (%)	Expected Survival to age 75 years (%)	Life Expectancy Gains in years
No Reduction	64.10	78.7	56.42	
Reduction to 60 $\mu g/m^3$	66.05	81.8	62.10	1.95
Reduction to 50 $\mu g/m^3$	66.71	82.6	63.5	2.61
Reduction to Background level	67.69	84.1	66.4	3.59

Table 3.20: 1995 Birth Cohort, Females

Pollution Reduction Scenario	Life Expectancy In years	Expected Survival to age 65 years (%)	Expected Survival to age 75 years (%)	Life Expectancy Gains in years
No Reduction	71.77	88.13	70.29	
Reduction to 60 $\mu g/m^3$	73.44	89.93	74.4	1.67
Reduction to 50 $\mu g/m^3$	73.94	90.39	75.5	2.16
Reduction to Background level	74.78	91.26	77.55	3.00

B. Population Alive in 1995

As described in Chapter 2, a phase in delayed effects scenario is introduced in the population alive in 1995 group. In this phase-in delayed effects scenario, slight changes are already introduced sometime before the tenth year for which the effects are delayed or whatever the specified number of years being studied, until a full effect takes place on the tenth year or the specified number of years. In this case, the phase-in of the effects was made on the fifth year for the ten-year delay, tenth year for the twenty-year delay etc.

For this population alive in 1995, 5-year life year gains per 100,000 population for different delay to full effects scenarios yielded a decreasing trend. To illustrate this, a reduction scenario to 60 $\mu g/m^3$ is used in Tables 3.21 and 3.22. Also, single age groups instead of 5-year age groups are used in these calculations so that the figures obtained here are slightly different from the previous figures.

It would be noticed from the numbers below that the longer the delay in the effects of the control measures, the life years gained for this population in the next five years also become less. Similar results are obtained with regard deaths that could be avoided in the next five years. Fewer deaths are avoided with the longer delay scenarios. The less than 65 years olds seem to benefit the most compared to the older age groups. Similar effects are seen in both males and females.

Table 3.21: Population Alive in 1995, Males:

Years Delayed	0	10	20	30	40
Reduction in Deaths w/in 5 years	Reduction in Deaths w/in 5 years	Reduction in Deaths w/in 5 years	Reduction in Deaths w/in 5 years	Reduction in Deaths w/in 5 years	Reduction in Deaths w/in 5 years
<65 yrs old	10,808	4,049	2,302	1,719	1,409
65-74 yrs old	3,254	1,150	588	400	301
>/=75 yrs old	2,261	817	418	285	216
Total	16,323	6,016	3,308	2,405	1,926
Life Years Gained w/in 5 years	75,720	45,606	39,728	37,785	36,758

Phased In Delayed Effects Gains in the first 5 years. Reduction to 60 $\mu g/m^3$ PM_{10}

Table 3.22: Population Alive in 1995, Females:

Years Delayed	0	10	20	30	40
Reduction in Deaths w/in 5 years	Reduction in Deaths w/in 5 years	Reduction in Deaths w/in 5 years	Reduction in Deaths w/in 5 years	Reduction in Deaths w/in 5 years	Reduction in Deaths w/in 5 years
<65 yrs old	5,690	2,183	1,279	977	819
65-74 yrs old	2,595	909	465	316	238
>/=75 yrs old	3,130	1,122	572	389	293
Total	11,414	4,213	2,316	1,682	1,350
Life Years Gained w/in 5 years	35,612	10,964	6,378	4,866	4,071

Phase in Delayed Effects Gains in the first 5 years. Reduction to 60 $\mu g/m^3$ PM_{10}

3.3.5. Results: Life Expectancy by City within Metropolitan Manila

From the data presented in the exposure assessment chapter, there are substantial contrasts of exposure for the whole metropolis from north to south. As seen from the average estimates by cities within Metropolitan Manila, a north to south exposure gradient exists. As part of this study including variability of effects within the metropolis, estimates of the predicted gains in life expectancy in a pollution reduction scenario for some cities with complete data are presented in this section.

As shown in Tables 3.23 and 3.24, reducing the pollution level to the national guideline for PM_{10} would give a range of differences by city in comparison with the no pollution reduction scenario of a little more than a month to as much as five years for the males and up to more than three years for females. The cities with higher pollution levels benefit the most from the reduction with their differences in life expectancies so much higher in cleaner air. Although the less polluted cities benefit as well, the differences in life expectancies are not as considerable as that of the more polluted ones. The exposure level of each

city is a primary contributory factor in the differences seen in the predicted gains in life expectancy among the different cities. Identical relative risk coefficients per 10 $\mu g/m^3$ PM_{10} were applied in all the cities. However, these coefficients vary according to the exposure level of the city and the level to which the PM_{10} is reduced, in this case, 60 $\mu g/m^3$ PM_{10}.

Another factor that could have affected the differences in the predicted gains in life expectancy among the cities in the given pollution reduction scenario is the pattern of baseline age-specific death rates of the populations among the cities. Some cities, for example, Valenzuela and Malabon have much higher infant mortality rates than others like Las Piñas. As seen earlier in the chapter, the application of a relative risk coefficient to modify the infant mortality could contribute substantially to the calculation of life expectancy. In cities with high infant mortality rates and high exposure levels of PM_{10} like Valenzuela and Malabon, modification of the rates in this age group in a pollution reduction scenario would yield higher predicted gains in life expectancy than in cities with lower infant mortality rates and lower exposure levels like Las Piñas. As for the adult age groups, a similar situation has been shown in Table 3.17 where a similar coefficient was applied in all three socio-economic groups with different patterns of baseline rates and yet, although little, differences were seen in the gains in life expectancy among the three groups. However as can be seen from Figures 3.8 and 3.9, it must be noted that the differences in the levels of exposure of the cities remain the major factor in explaining the differences in the predicted gains in life expectancy in the pollution reduction scenario.

***Table 3.23**: Comparison of the 1995 Life Expectancies among Males in Cities of Metropolitan Manila considering Two PM_{10} Pollution Scenarios*

City	No Pollution Reduction	Pollution Reduction to 60 $\mu g/m^3$	Predicted Number of Years Gained	PM_{10} levels, $\mu g/m^3$
Valenzuela	66.19	71.25	5.06	153
Malabon	66.24	71.06	4.82	146
Caloocan	65.22	69.59	4.37	137
Quezon	66.81	70.37	3.56	124
San Juan	67.89	70.98	3.09	117
Mandaluyong	66.60	69.67	3.07	113
Manila	62.58	65.58	3.00	109
Makati	67.86	69.94	2.08	99
Taguig	66.36	67.90	1.54	87
Pasay	66.74	68.00	1.26	81
Parañaque	62.34	63.00	0.66	71
Las Piñas	69.92	70.02	0.10	66

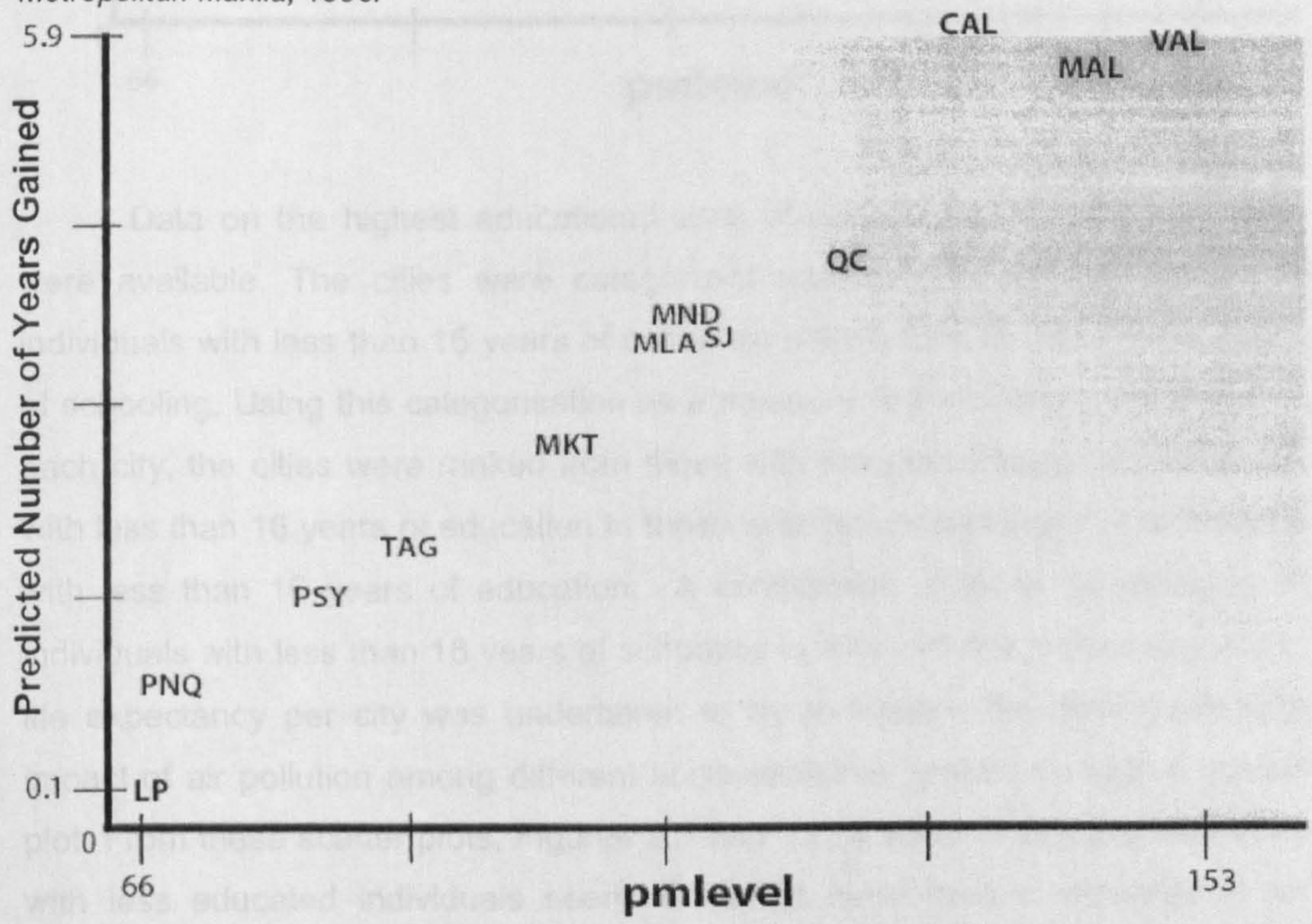
Comparison shows selected Cities in Metropolitan Manila ranked according to PM_{10} levels and life expectancy in years among Males, 1995

Table 3.24: *Comparison of the 1995 Life Expectancies among Females in Cities of Metropolitan Manila considering Two PM_{10} Pollution Scenarios*

City	No Pollution Reduction	Pollution Reduction to 60 $\mu g/m^3$	Predicted Number of Years Gained	PM_{10} levels, $\mu g/m^3$
Valenzuela	71.46	75.15	3.69	153
Malabon	70.84	74.33	3.49	146
Caloocan	70.45	73.70	3.25	137
Quezon	71.57	74.19	2.62	124
San Juan	70.58	73.07	2.49	117
Mandaluyong	70.44	72.83	2.39	113
Manila	67.85	70.24	2.39	109
Makati	72.08	73.72	1.64	99
Taguig	71.36	72.60	1.24	87
Pasay	71.72	72.73	1.01	81
Parañaque	67.41	68.01	0.60	71
Las Piñas	73.46	73.82	0.36	66

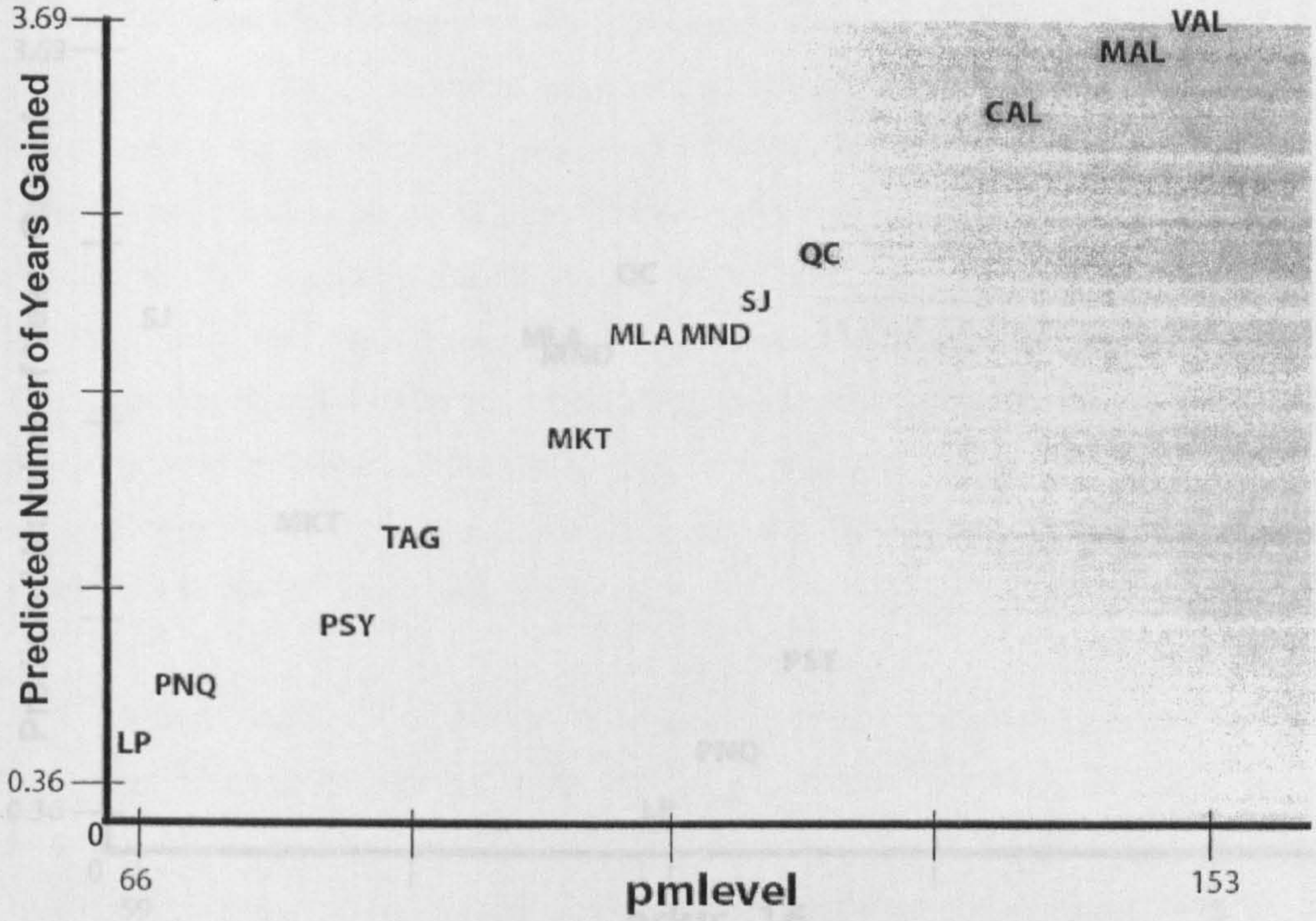
Comparison shows selected Cities in Metropolitan Manila ranked according to PM_{10} levels and life expectancy in years among Females, 1995

Figure no. 3.3. *Predicted gains in Life Expectancy vs. the PM_{10} Level in $\mu g/m^3$, among males, Metropolitan Manila, 1995.*



Legend: LP - Las Piñas; PNQ – Parañaque; PSY – Pasay City; TAG – Taguig; MKT – Makati City; MLA – Manila; MND – Mandaluyong; SJ – San Juan; QC – Quezon City; CAL – Caloocan City; MAL – Malabon; VAL - Valenzuela

Figure no. 3.4: *Predicted gains in Life Expectancy vs. the PM_{10} Level in $\mu g/m^3$ among females, Metropolitan Manila, 1995.*



Data on the highest educational level of residents in the different cities were available. The cities were categorised according to the percentage of individuals with less than 16 years of schooling and those with 16 or more years of schooling. Using this categorisation as a measure of socio-economic status of each city, the cities were ranked from those with low percentages of individuals with less than 16 years of education to those with high percentages of individuals with less than 16 years of education. A comparison of these percentages of individuals with less than 16 years of schooling by city with the predicted gains in life expectancy per city was undertaken to try to explore the disproportionate impact of air pollution among different socio-economic groups through a scatter plot. From these scatter plots, Figures 3.5 and 3.6, it seem to suggest that cities with less educated individuals seem to benefit more from a reduction in air pollution than cities with more educated individuals, however, it is clearly not a strong relationship especially for the females. Also, this suggests that the cities where the less educated reside are the more polluted ones. However, it must be noted that this ecological comparison is crude and suffers from several limitations such as effect of lifestyle factors.

Figure no. 3.5: *Predicted gains in life expectancy vs. Percentage of individuals with 16 years of education among females, Metropolitan Manila, 1995.*

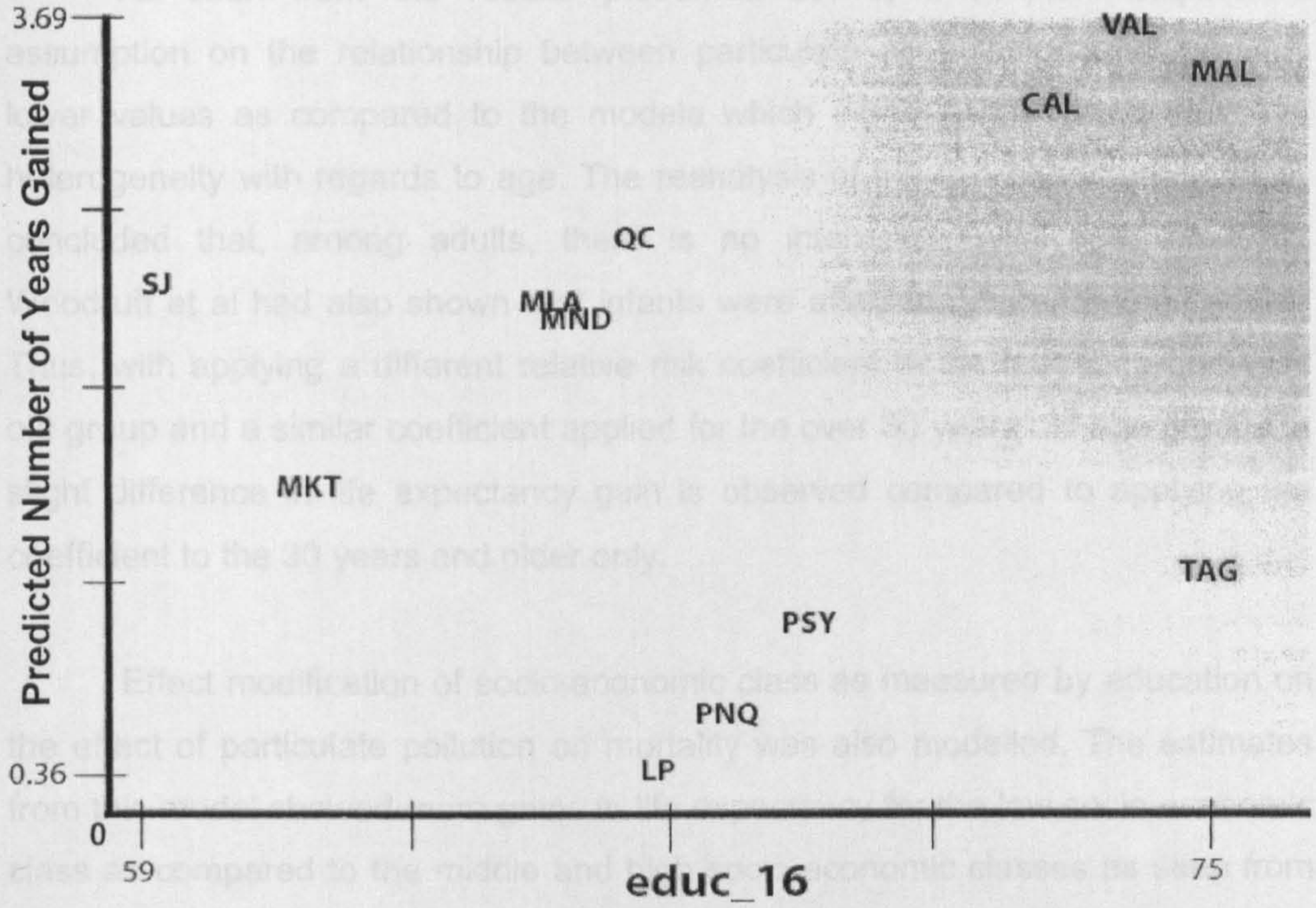
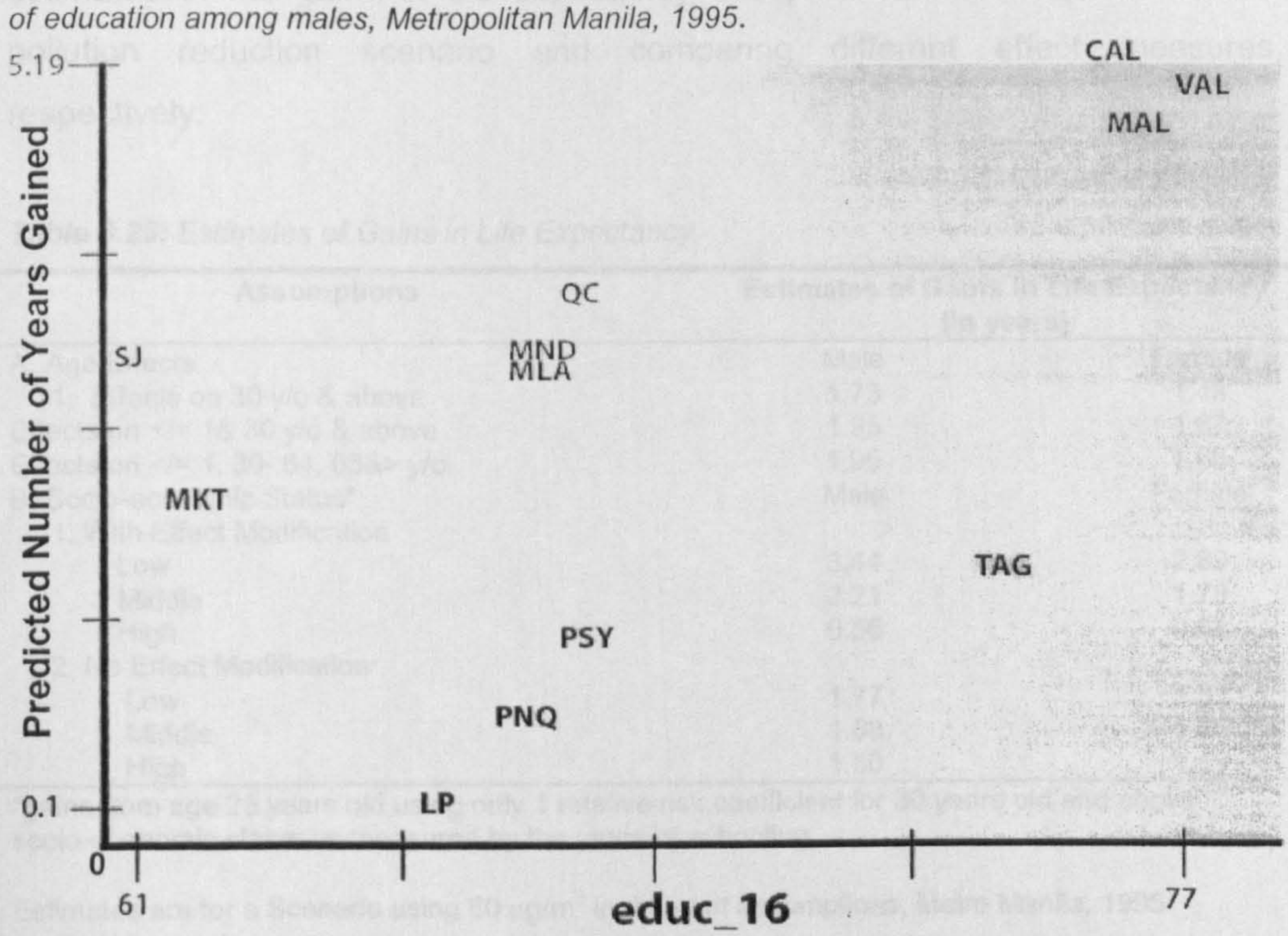


Figure no. 3.6: *Predicted gains in life expectancy vs. Percentage of individuals with 16 years of education among males, Metropolitan Manila, 1995.*



3.3.6 Complex Models: A Synthesis

As seen from the results presented above, a simple multiplicative assumption on the relationship between particulate air pollution and mortality lower values as compared to the models which introduced a certain level of heterogeneity with regards to age. The reanalysis of the two cohort studies had concluded that, among adults, there is no interaction with age; however, Woodruff et al had also shown that infants were affected differently than adults. Thus, with applying a different relative risk coefficient to the less than one year old group and a similar coefficient applied for the over 30 years old age groups, a slight difference in life expectancy gain is observed compared to applying the coefficient to the 30 years and older only.

Effect modification of socio-economic class as measured by education on the effect of particulate pollution on mortality was also modelled. The estimates from this model showed more gains in life expectancy for the low socio-economic class as compared to the middle and high socio-economic classes as seen from different pollution reduction scenarios. The following two tables summarize the estimates of the gains in life expectancy using different assumptions in one pollution reduction scenario and comparing different effect measures, respectively:

Table 3.25: *Estimates of Gains in Life Expectancy*

Assumptions	Estimates of Gains in Life Expectancy (in years)	
A. Age Effects	Male	Female
1. Effects on 30 y/o & above	1 73	1.48
Effects on </= 1& 30 y/o & above	1.95	1.67
Effects on </= 1, 30- 64, 65&> y/o	1.95	1.68
B. Socio-economic Status*	Male	Female
1. With Effect Modification		
Low	3.44	2.89
Middle	2.21	1.78
High	0.56	0.45
2. No Effect Modification		
Low	1.77	1.51
Middle	1.68	1.36
High	1.50	1.19

*gains from age 25 years old using only 1 relative risk coefficient for 30 years old and above, socio-economic status as measured by the years of schooling.

Estimates are for a Scenario using 60 $\mu g/m^3$ in different assumptions, Metro Manila, 1995

Table 3.26: Different Effect Measures

Effect Measure	Male	Difference	Female	Difference
1. Life Expectancy (years)				
a. No Pollution Reduction	64.10		71.77	
b. With Pollution Reduction	66.05	1.95	73.44	1.67
2. No. of Deaths w/in 5 years: Population Alive in 1995				
a. No Pollution Reduction	135,437		90,978	
b. With Pollution Reduction	119,114	16,323	79,564	11,414
3. Life Years w/in 5 years: Population Alive in 1995				
a. No Pollution Reduction	404,596		278,830	
b. With Pollution Reduction	328,876	75,720	243,218	35,612

Effect Measures are for the Estimates of Gains in a Scenario using 60 $\mu g/m^3$, Metro Manila, 1995

Chapter 4

DISCUSSION, CONCLUSIONS AND IMPLICATIONS

Before discussing the results found in this impact assessment, this chapter addresses some issues regarding the process of risk assessment, the data that was used and its analysis, the uncertainties inherent in risk assessments, and in particular the direction by which such uncertainties would affect the results. This chapter thus discusses the potential for bias in the data used, the problems in exposure assessment common among air pollution studies, and the assumptions of the models: heterogeneity, cardio-pulmonary causes of death and the delayed effects are deliberated. The public health impact of the methodology and the results including reactions of policy makers are likewise listed and described. Finally, the general conclusions and the study's implications are enumerated. Discussion of the results of the cities within Metropolitan Manila has been incorporated in section 3.3.6, Chapter 3, as it was easier to describe the findings by discussing it as well.

4.1. The Risk Assessment Process and the Uncertainties

Risk assessment is a method that predicts human health risk in circumstance where risk cannot be measured directly. In risk assessment the data on the effects on animals or a particular population are used to predict the effects on another human population, exposure of who are generally different from the original studies. Animal studies have been used quite extensively in a number of risk assessment exercises for different toxic substances. Of late, epidemiological studies have begun to be used for such purpose (*Hertz-Picciotto, 1995*).

Regulatory agencies make use of risk assessment for the purpose of setting priorities for both their own agenda and research and for developing standards. Non-regulatory agencies such as labour unions, industries, consumers and the general

community also make use of this method. The latter's functions are varied, e.g. monitoring and surveillance in occupational settings and for consumers' protection. (*Samet et. al., 1998*) In this present study, the functions defined are not only for regulatory purposes, i.e. setting standards, but also to assess the needs of specific programmes that would meet the needs of specific parts of the population who might be vulnerable to the environmental pollutant under study. More importantly, risk assessment can provide guidance on the benefits of implementing mitigation measures that would take into account health through the economic valuation technique. These will be discussed more thoroughly in the public health impact section.

Risk assessment studies involving air pollution that has been carried out have been done mostly in developed countries (*Hurley et. al., 2000; Brunekreef et. al., 1997; Nevalainen et. al., 1998; Kunzli et. al., 2000*). In the case of developing countries, the study that was been done in Santiago, Chile, where mortality reduction was predicted using the country's annual average guideline/standard. However, the exposure-response estimates used were a blend of a pooled estimate of time series studies and the cohort study. The study utilized the time series coefficient as the low estimate, the cohort coefficient as the high estimate and a central estimate derived from the arithmetic mean of the low and high coefficients (*Ostro 1996*). In the recent study undertaken in three cities of developing countries including Santiago, Chile, Sao Paolo, Brazil and Mexico City and one city from a developed country, New York City, the exposure-response coefficients in the health assessment also made use of both the cohort and time series studies. This time the low estimates were from the time series studies conducted in the local areas and the central and high estimates were based on the cohort studies. The authors argued that the time series studies did not reflect life shortening of only a few days or weeks as argued in a recent analysis, thus, their use in risk estimations was useful (*Cifuentes et. al., 2001*).

An important source of uncertainty in the present study is the transferability of the exposure response coefficients from the cohort studies to the Metropolitan Manila population. Unlike the risk assessment studies done by Kunzli and Hurley et

al, the transfer of the coefficients from the US to the Philippines is more complicated. The Philippines is a developing country where the levels of exposure of particulates in the population, general socio-economic conditions, pollution sources and mix, and mortality data are different from that of the United States. Nevertheless, after a thorough discussion of the issues and elaboration of the strengths and weaknesses of such adaptation, the use of the cohort studies' coefficients was deemed appropriate.

An added uncertainty about transferability of coefficients is on the socio-economic mortality coefficients. As explained in Chapter 3, part of the study would like to explore possible or likely differential effects among different socio-economic classes. Unfortunately, no data were available in the Philippines on deaths by socio-economic classes. Therefore, coefficients from the US were adapted. It is recognized that this is a limitation of the study. Opting to use the US data tended to underestimate the real differences in adult mortality among socio-economic classes considering that a wider socio-economic gap is generally seen within developing countries. Thus in the interpretation of the results regarding the effects of particulate pollution among socio-economic classes, this caveat must be considered.

Two other elaborations were applied in this study. The first study used data from the infant study by Woodruff and the age interactions contained in the time series studies, and also investigated age effect. Secondly, the differential impacts of effects of particulate pollution differ among socio-economic classes. These two issues will be discussed further in the results section of this chapter. The uncertainty on the exposure assessment i.e. using fixed sites monitors to represent the population's exposure level is also discussed in the section on information bias.

To deal with the uncertainties, Kunzli recommends a prudent approach in the estimation of the attributable deaths or cases by using an 'at least' principle. This approach in case of mortality involved the non-inclusion of the potential effects of particulate pollution on infants, resulting in conservative estimates. While this approach is commendable, however, in the present study, we go a step further. Different pollution reduction scenarios and different assumptions regarding the age

and socio-economic effects were explored. In this process, the scenario similar to Kunzli's recommendation was also investigated. Comparison of these scenarios made it possible to test the sensitivity of the methodology and results to the different assumptions. Like Kunzli, however, the results in this present study are interpreted with caution.

In spite of the presence of a number of uncertainties in this type of approach, it remains quite beneficial to employ this type of approach for a couple of important reasons as Kunzli succinctly explains. It is recognized that ambient air pollution, specifically particulates, has adverse health effects including death. Therefore, there is some public health impact that should be estimated and made known to the public. Secondly, and as has been discussed earlier, policy decisions and standard setting must take into consideration public health. Despite the uncertainties of this technique, risk assessment of this type is considered to be very useful in incorporating the impact of certain policy decisions or programmes, especially those environmental ones, on health. (*Kunzli et. al., 2000*)

4.2. Errors in Baseline Death Rates

In using secondary data, i.e. vital statistics or routinely collected data, the completeness and accuracy of the data set could be a concern. Secondary data are readily available and relatively inexpensive. In this and succeeding sections, these concerns are discussed and dealt with accordingly.

In constructing a life table for this present study, age-specific mortality rates were used. The National Statistics Office for Metropolitan Manila[1] provided these data. As mentioned in Chapter 2, a detailed and careful study of the death registration in the Philippines showed that under-registration of deaths within the metropolis is negligible. In the study, estimated deaths for the metropolis were compared with those who were actually registered. The results suggested that the level of unregistered deaths was only 0.6% (*Flieger et. al., 1992*). Deaths of residents

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[1] This office is in charge of gathering, analysing, and encoding all death certificates from different city governments within the metropolis.

who lived in Metropolitan Manila but died outside the metropolis[2] are registered in the place where it occurred. This is estimated to be about 4-5%. However, what the National Statistics Office does is collate all the death certificates from various parts of the country and reassign deaths to the area where people lived prior to death. (*Espinoza, 2000*) Therefore, the completeness and accuracy of the data set in this study could not have affected the estimates grossly.

4.3. Errors in Exposure Estimates

Routinely collected exposure-monitoring data on particulate matter and levels of education of the Metropolitan Manila population from the 1995 census were used in this study. Inaccuracies regarding information on causes of death and/or place of residence of the deceased, monthly or annual averages of the levels of particulates in the eight monitoring stations, and categorization or reporting of the levels of education in the census could lead to random error. This is known as information or misclassification bias. This type of bias is connected with problems in the instruments and techniques of data collection, hence compromising the information gathered on exposure, disease or the other study variables (*CRC Press, 1995*).

This section describes the collected data based on its completeness and accuracy. Some methodological issues about the collection of these data are and the exposure assessment process, which is crucial in this endeavour, is likewise reviewed.

As discussed in section 4.2, the data set on mortality based on the death registration system is almost complete and under-registration is negligible. Therefore completeness is not an issue. A more daunting aspect of the death registration system is the classification of the cause of death. Although all deaths were considered in this study, a section of the results pertains to those deaths caused by cardio-respiratory conditions. Hence, consideration of the classification of the causes of death also became important.

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[2] Most of these deaths were due to violence or traffic accidents.

Based on the system for death registration, all causes of death were provided by medically qualified personnel, i.e. attending physicians and/or the municipal health officers. The proportion of deaths with ill-defined causes was about 3.4%. In such cases, the Vital Registration Division of the NSO, which is responsible for the death registration system, would check with the hospital where the certificate originated or contact the attending physician and/or the municipal health officer for clarification (*Espinoza, 2000*). As a result of this process the classification of the causes of death was minimized.

Another set of data that was used in this study was the classification of the level of education of Metropolitan Manila. The data was collected through the census done in 1995. Data collection required interviewers to ask the household head the details about the members of the household, which, it was anticipated, could result in a problem of recall bias. There are no local studies that looked into this occurrence. Thus, added to the uncertainty of the allocation of deaths by the different educational groups, as discussed earlier, interpreting the results about the differences of effects among different socio-economic classes must be done with caution. It must be remembered that presenting those results was primarily just to show probable differences among certain sub-populations that are generally considered vulnerable.

Exposure assessment is considered to be one of the most difficult tasks (*Rothman K., 1998*). In the case of ambient air pollution studies, assignment of levels of exposure of individuals is usually based on a single fixed monitoring station (time series studies) or a network of monitoring stations. In the latter case, values from the different stations are averaged and this average is used to represent the level of exposure of individuals in that particular area.

In this assessment, annual average of PM_{10} for 1995 was utilized to represent the level of exposure for the whole of Metropolitan Manila. This annual average was a combination of readings from the eight monitoring stations throughout the said year. Averaging across these stations and through time indicated that the variability in levels of particulate pollution across different areas and time was not considered.

Hence, the estimated attributable deaths or loss in life expectancy could have been underestimated. Even the attempt to assign exposure levels through the use of GIS extrapolation from the available data and data points would likewise result to some underestimation of the attributable deaths or loss in life expectancy. However, the method of averaging the values minimizes the problems in assigning levels of exposure of those individuals who move around a lot and are, therefore exposed to different levels of pollutants most of the time. (*Gouveia, 1997*)

The representativeness of the ambient measurements of an individual's 24-hr exposure is an issue in air pollution studies because most individuals spend their time indoors where levels are expected to be lower unless sources, i.e. smoking, type of cooking fuel, are present. However, in a tropical country like the Philippines, it is common for people to have windows that are fairly large and usually remain open for most of the time. Such practice would therefore presume little difference between ambient and indoor levels. Thus, in this assessment, using the ambient levels as a substitute for the population's exposure is more justifiable.

In summary, problems with the classification and completeness of the mortality data set were greatly reduced by the process by which the data where checked and collected. The estimates in the results using education classification from the census data, with the potential for recall bias and together with the uncertainty of the transfer of US mortality ratios, must be carefully interpreted, since some degree of misclassification could have occurred. The estimates, however, could be expected to be conservative or in agreement with the 'at least' approach, and prudent which is desired for this type of assessment (*Kunzli et. al., 2000*).

4.4. Results

4.4.1. Base Multiplicative Model

In this study, the base multiplicative model, which used single relative risk applied to adults 30 years and over, found that health gains in life expectancy when reduced to the international guideline of 50 $\mu g/m^3$ PM_{10} would be approximately 2 years for both men and women, the former a little more and the latter, a slightly less.

The reduction was 45.6 µg/m3 from the exposure of 95.6 µg/m^3 PM_{10}. Using other reduction scenarios such as the national guideline and total lowering to background levels yielded health gains in life expectancy proportional to the said reduction, 35.6 µg/m^3 and 66.35 µg/m^3 PM_{10} respectively.

In England and Wales, the estimated gain in life expectancy is just under 5 months for every 10 µg/m^3 decrease in PM_{10} with a range of about 10 to 28 weeks (*Hurley, 2000*). For this study, when converted to decreases of 10 µg/m^3, the gain in life expectancy is about five and a half months for males and a little over five months for females. These results are thus consistent with the estimates in England and Wales. The slightly higher gains in this study are due to the use of a slightly higher relative risk. The relative risk used here is a pooled estimate from both the Six-Cities and ACS studies, i.e. 1.05 (1.03-1.07), whereas the IOM report used only the relative risk in the ACS study of 1.04, which is slightly lower.

***Table 4.1**: Life Expectancy Differences from Two Pollution Scenarios, Metropolitan Manila, 1995.*

Pollution Scenario	MALE	Difference	FEMALE	Difference
No Reduction	64.1		71.77	
Reduction by 10 µg/m3	64.57 (64.4-64.77)	0.47 (0.3-0.67)	72.19 (72.03-72.36)	0.42 (0.26-0.59)

The size of the effect of reducing particulate pollution by 10 g/m3 in Brunekreef's paper on life expectancy gains among Dutch adult males is about 1.11 years. His result is much bigger than seen in this study. This was a function of the relative risk used for the reduction, which was 1.10 per 10 µg/m^3. The relative risk coefficient used by Brunekreef is an overestimate of the Six-Cities and ACS studies' relative risks. Thus, the big difference between our results is observed. Since the gains in life expectancy utilising these similar methods are sensitive to the coefficient used, Brunekreef's results are understandably greater. (*Brunekreef, 1997*)

Nevalainen et al, likewise, investigated the effect of particulate pollution on life expectancy. Their study revealed similar results with the Study on England and Wales and this study. Using the ACS coefficient, which, in their calculation is slightly higher than the England and Wales study at 1.07, the gain in life expectancy was 0.6

years or 6 months for the whole population. Nevertheless, the Nevalainen's result is still within the range of the results reported in this study (*Nevalainen, 1998*). In addition, similar gains in life expectancy, approximately 6 months per 10 $\mu g/m^3$ PM_{10}, was cited by Kunzli et al in a study conducted on estimating the attributable cases due to air pollution for three countries, namely Austria, France and Switzerland. (*Kunzli, 2000*)

The assessments undertaken in the developing countries in the east such that made in Bangkok and New Delhi were based mainly on time series studies and not on cohort studies as had been in the studies cited above. Thus, the results of this study and the studies from the east are not comparable.

Summarising the above, it could be surmised that using the base multiplicative model in this study yielded results comparable with the results of other studies cited. However, due to the sensitivity of these results to the coefficients used, slight differences exist. In addition, the amount of exposure of the population also affects the results as seen from the different pollution reduction scenarios.

4.4.2. Heterogeneity

As discussed earlier in Chapter 3, there is evidence from the literature that particulate pollution could affect infant mortality *independently from adult mortality (Woodruff et. al., 1997; Bobak et. al., 1999*). This evidence was likewise appreciated by Kunzli et al, but chose to ignore these potential effects on infants since infant mortality in the countries of concern were relatively low and therefore the attributable cases would be low as well. However, they pointed out the importance of including this evidence on infants for economic valuation purposes. (*Kunzli, 2000*) The implication on life expectancy and the years of life lost could be substantial especially in countries with high infant mortality rates like the Philippines. Comparing the life expectancies for the different pollution reduction scenarios, which included the infant exposure-response coefficient shown in Table 3.12, with that which does not, as shown in Tables 3.8 and 3.9, resulted in differences of about 0.22 to 0.40 years for males and 0.19 to 0.34 years for females. Translating these to the number

of months, that would be 2 and half months to less than five months for males and 2 to 4 months for females. In terms of years of life lost over the number of people, this could indeed be quite sizeable and would have greater implications in economic valuation as rightfully suggested by Kunzli et al.

Apart from this, the time series studies have likewise some indication that the magnitude of effect among the elderly population is slightly higher than the general population and/or those younger than 65 years old. (*Schwartz, 1994; Saldiva et. al., 1995; Ponka et. al., 1998; Cropper et. al., 1997; Chestnut et. al., 1998*) In addition, as will be discussed later, the plausibility of the biological mechanism as regards the effects of particulate pollution on the most vulnerable, such as the aged, supports the notion of a slightly different effect among the elderly. However, in the reanalysis of the ACS and Six-City studies, no interaction with age was apparent (*Krewski et. al., 2000*). Nevertheless, this study explored this possibility of variability with the elderly, although slight, to demonstrate the effect of integrating risks among the vulnerable part of the population on the overall life expectancy. As was seen from the results, when a third exposure-response coefficient for ages 65 years and over was included, the gains in life expectancy remain almost similar for males and slightly higher for females compared to the model using coefficients for infants and for 30 years and over alone. As explained in the previous chapter, this occurrence is probably due to the differences in the baseline mortality rates among the males and females.

Exploring the effects of the variability of age, unfortunately, has not been made in other risk assessment exercises so that comparison with this study's results could not be done. Risk assessments done in cities of the developing world such as those in Bangkok and Jakarta, included effects on general population using coefficients from time series and/or cross-sectional studies done in the early 1990's without assessing the effects of age (*WB, 1994; USAID, 1990*). The same is true with the health impact assessment conducted in the Philippines some years back (*WB, 1996*). The risk assessments done in the UK, Finland, Netherlands, Austria, Switzerland and France, which used the coefficients from the cohort studies, did not

investigate the effects of age in coming up with attributable cases or attributable years of life lost (*Brunekreef et. al., 1997; Nevalainen et. al., 1998; Hurley et. al., 2000; Kunzli et. al., 2000*).

In the literature review, it has generally been shown that social class, using any or all of the indicators discussed, is closely related to differences in mortality. However, there has been no study which could show quite clearly that the combined effects of socio-economic class and particulate pollution would have deleterious effects until the results of the reanalysis of the Six-Cities and ACS studies came out. Although, evidence of differential exposures to outdoor concentrations of the different socio-economic classes and/or racial groups had been previously reviewed (*Sexton et. al., 1993*), it is only with the ACS and Six-City studies reanalysis that the combined effects of socio-economic class and exposure to particulate pollution on mortality had been clearly evident. Zanobetti and Schwartz likewise demonstrated the effect modification brought about by socio-economic class on particulate pollution and mortality in a time series study in the four largest US cities but rather weakly (*Zanobetti et. al., 2000*).

As part of this present study, both the interaction between socio-economic class and particulate pollution and no interaction have been considered. The basic model investigated the assumption that particulate pollution affects different socio-economic classes as measured by education equally. From the results, the gain in life expectancy among 25 years olds is seen among the low socio-economic classes for both males and female as well as for the middle and the high socio-economic class. However, this relationship between particulate pollution and mortality within different socio-economic classes has a more complex dimension. Evidence from the ACS and Six-City reanalysis has shown that particulate pollution affected mortality in different socio-economic classes differentially. As applied in this data set, the findings revealed results similar to those of the simpler model but the magnitude has increased for two of the three socio-economic classes. With the interaction terms from the ACS reanalysis, the gains in life expectancies in the three categories would

increase quite considerably for both the low and middle socio-economic classes as compared to the basic model but not for the high socio-economic class.

In considering which is the more appropriate model for the Metropolitan Manila population, there is a tendency to favour the more complex model because of the reason stated above (evidence of interaction in the cohort studies). But both models have their own merits. The basic model has a smaller number of assumptions about transferability. The more complex one does not only require the assumption about the transferability of risk from one population to the other but also of the transferability of the quantitative estimate of the interaction. And so, it is reasonable to infer that it cannot be certain as to which gives the better estimate between the two models but both nevertheless give an idea of the range or extent to which these estimates are vulnerable to the particular assumptions about different vulnerability. Thus it could be said that the gains in life expectancy among the low socio-economic for males for a reduction to 50 µg/m3 PM_{10} could be from 2.33 years to 4.37 years, 2.21-2.8 years for middle socio-economic class, and 0.74 – 1.97 years for high socio-economic class. For females, it would be, for the same reduction scenario, 1.98-3.63 years for low socio-economic class, 1.78 – 2.24 years for middle socio-economic class and 0.59 – 1.57 years for high socio-economic class.

As in the age variable, this aspect was not explored by any of the risk assessment studies that have so far been done. Nevertheless, these differences of life expectancies among the socio-economic classes attributable to particulate pollution are consistent with other studies, which did not necessarily use mortality or life expectancy but other health outcomes of environmental pollution. In a review by Brown about the role of race, class and environmental health, he concluded that 'the overwhelming bulk of evidence supports the "environmental justice" position that environmental hazards resulting to adverse health effects are inequitably distributed by class and especially race' (*Brown Race, 1995*). Local studies in the Philippines on environmental pollution such as water contamination problems resulting to gastro-intestinal diseases, air pollution problems resulting to higher levels of respiratory

diseases and chemical contamination leading to poisoning, support this view (*WB, 1996*).

In summary, in considering the effects of age, particularly among infants, when assessing the attributable years of life lost due to particulate pollution could be potentially important as substantial differences are seen compared to that without taking this variable into account. Exploring the effects on different socio-economic classes as indicated by education, also gives an insight as to the differences in the effects on more vulnerable sub-population in Metropolitan Manila.

4.4.3. Probable Biological Mechanisms of the Interaction

Judging from the evidence presented in the literature review regarding the probable biological mechanisms underlying the relationship between particulates and mortality, these mechanisms are rendered more fatal when certain vulnerabilities are present. Furthermore, it has been shown by the animal experiments that the presence of a disease process such as the coronary occlusion in the dog study, that the health effect for those exposed to combined ambient particles, in this case ischemia, started so much sooner than those with sham exposure (*Godelski et. al., 2000*). Other experiments on rats and mice have also revealed similar results (*Gavett et. al., 1999; Brown Race, 1995; Watkinson et. al., 1998*).

And so, are the effects of particulate pollution only added on to individual vulnerability? In my opinion and in the absence of a mathematical model that can establish these events more clearly, the interaction between the particulate effect and the individual vulnerability is probably multiplicative in nature rather than additive. The mortality risk is restricted to those who are already compromised and the effect on those who are not compromised was not considered to be extensive nor severe. Thus, this goes beyond just the additivity of the effects. In fact, in a related animal experiment as discussed earlier, although the endpoint was not fatality, the effects seen were more than additive in nature. Interaction between particulate matter and sensitised mice resulted in three to eight fold increases in eosinophils and inflammatory agents (*Gavett, 1999*). In the same way that diseased

individuals are susceptible, as one ages, the human body gets to be more vulnerable to insults, which have accumulated due to exposure to all sorts of hazards throughout the years or just the wear and tear brought about by aging itself. In addition, there is the particulate pollution factor[3]. With the combination of these two factors, age and particulates, the decrease in lung function and other effects are considerable and probably multiple. Thus, in elderly individuals, the effect of particulate pollution is compounded and would become more pronounced leading to mortality.

4.4.4. Modifying only the Cardio-Pulmonary Deaths

The gains in life expectancies in modifying the cardio-pulmonary causes of death are quite considerable. As mentioned in the results chapter, majority of the causes of death in the attributable fraction is cardio-pulmonary in origin. Time series studies that had investigated cardiovascular mortality and particulate pollution had consistently found a positive association, some with higher relative risks than all cause mortality, with increases in particulates. (*Ballester et. al., 1996; Ponka et. al., 1998; Zmirou et. al., 1998*) Pope summarised several of these studies to show this occurrence. He showed that 69% of excess deaths due to particulate matter exposure were due to cardiovascular disease and 28% were due to respiratory disease. Only 3% of the excess deaths were due to other diseases (*Pope, 2000*). Furthermore, the plausible biological mechanisms involved here, as discussed earlier, are primarily cardio-pulmonary in nature. Thus, it could be inferred that in modifying the cardio-pulmonary causes of death attributable to particulate pollution, an impact on the life expectancies could be demonstrated.

4.4.5. 1995 Birth Cohort and Population Alive

The aim of this part of the results and discussion is to speculate on other scenarios and explore different effects measures, which can help policy makers in their decision making. The scenarios presented in this section involve delayed effects for the population alive in 1995. The actual length of delay before effects

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[3] In fact, as discussed in the literature review, lung function diminishes after age 20-30 years old. Diminished lung function leads to premature mortality. (*Evans et. al., 1997*)

take place is not known, of course. The scenarios assumed that birth, death and migration rates are constant throughout the time period of study. Thus, for example, the reduction of the number of deaths in the population alive in 1995 could be an overestimate. The report on England and Wales further explains that since the population size from 1995 would decline in time, the number of deaths would likewise decline even without the change in air pollution. Hence, the number of deaths attributable to air pollution would be less than indicated in the results. This might not be a gross overestimate when calculating within the 5-year period but it would be so if applied for longer periods of time (*Hurley et. al., 2000*). Nevertheless, the usefulness of such scenarios is fairly important especially when economic valuation could be done.

Apart from the gains in life expectancies, the scenarios also included other effect measures such as mortality reduction (number of deaths) and years of life lost within 5 years for the population living in 1995 and expected survival till a certain age for the 1995 birth cohort. Different ways of representing the gains in reducing particulate pollution could give different dimensions as well as some clarity in the understanding of such reduction. The number of deaths or mortality is the most commonly used effect measures in health studies. Presentation of such results using different effect measures yielded some interesting reactions from policy makers as will be discussed later in the chapter.

4.4.6. Public Health Impact

Health risk assessment, as mentioned in the literature review, is a risk management tool used extensively by regulatory officials and scientists alike, especially in developed countries. Since 1970 the field of risk assessment has grown tremendously (*Bates, 1994*). In the United States for instance, comparative risk assessments were used for prioritising environmental health programmes in both state and local health departments (*Johnson, 2000*). In developing countries, the extent of utilisation of such a method is not known. In the Philippines, it was only in the past five years that health risk assessment of development projects began to be

implemented, e.g. assessing potential health risk posed by coal-fired power plants, cement factories, establishment of industrial estates, to name a few.

A Philippine environmental health assessment that was undertaken in 1995 affirmed the health effects of air pollutants, which lent added urgency to the passing of the Clean Air Act. However, due to certain limitations in the project's methodology as discussed in the literature review, its usefulness in setting local standards or in formulating environmental health policies was practically ignored. Air quality standards, for example, in the Clean Air Act are copied from other countries' air quality guidelines without regard as to the practicability of its application locally. In the area of health, although general health programmes are in place, vulnerable populations have not been considered with regards to environmental exposures. The methodology and model used in the study could help in both instances. It estimates the public health impact of air pollution according to the level of reduction, and, at the same time, identifies and estimates effects on certain vulnerable groups. Therefore, in using the methodology and results of this exercise, policymakers can arrive at a more informed decision regarding air quality standards and health policy that are based on scientific evidence.

It must be noted further that the small effects seen in epidemiological studies on air pollution yielded quite substantial estimates of public health impact. These substantial estimates could be due to the fact that the whole population is exposed to air pollution unlike some risk factors such as lifestyle factors where population exposure could be a product of choice.

Taking into consideration the results and methodology of this research, the Annex 3 table shows the steps where these results can be most useful. This table shows a general framework for the control of air pollution with Metropolitan Manila. With the passage of the Clean Air Act, a policy instrument has been set into motion within the driving force stage. Actions in the other stages are either in progress or are soon to follow. However, there are implementation issues to be considered. Comments on the actions/policy instruments and their implementation are also elaborated upon in this table. The comments give the local context by which these

actions/policy instruments would have to be or are being adapted and implemented (*EMB-DENR, 2000; Heil, 1998*).

First of all, the results provide the economic basis for adopting different actions and policies. Using the life years gained from pollution reduction, a cost-benefit analysis can be done to justify adaptation of particular programmes or actions/policies at this stage. Secondly, the results can be used in air quality standard-setting. It is at this stage that the results and the methodology are most helpful. Since the review of the standards are made every 2 to 3 years, these research results and the methodology used here could become part and parcel of the decision making process in arriving at a standard which is more practicable, realistic and achievable locally at the present time. The basis of the standard could also be guided by differential impact among different vulnerable groups.

Finally, a progressively constructed health policy and/or programme could be formulated. The apparent north-south differences in levels of pollution seen in this study would also be useful for the local governments in those cities in terms of limiting factory operations or decreasing vehicular traffic within their specific areas.

4.5. General Conclusions and Implications

This health risk assessment was carried out to estimate the public health impact of air pollution as indicated by PM_{10} on mortality in Metropolitan Manila, Philippines. Using routinely collected data and the life table approach, the long-term effect on mortality was computed. Heterogeneous sub-populations were likewise explored. Crucial assumptions affecting validity such as the transferability of coefficients were carefully dealt with and discussed. In summary, the following conclusions can be drawn from this assessment:

1. Using mainly the coefficients from the mortality cohort studies in the USA as exposure –response relationship, aided by the mortality time series studies from the developed and primarily developing countries, and utilising life table methods, it is possible to estimate the effects of air

pollution with PM_{10} as indicator on total mortality in Metropolitan Manila, Philippines. This assessment has shown that routinely collected data, i.e. death registration system and air pollution measurements were valuable for the estimation of public health impact of air pollution. These data were accessible, although improvement in quality and amount of information collected is certainly encouraged. Routinely collected data is an important resource in developing countries in risk assessment. These data form the basis from which public health impact could be measured. In developing countries, where there is a lack of epidemiological studies to measure effects of air pollution directly, public health impact assessment using such routinely collected data is extremely beneficial for decision-makers.

2. There are uncertainties associated with estimating public health impact of air pollution. The main concern is the transferability of the US coefficients to a developing country like the Philippines. However, several studies, most of which were time series studies, have shown that the magnitude of effects of air pollution, at least for that design, was similar in many different countries, both developed and developing. Therefore, it is suggested that the transferability of the US coefficients to other countries for risk assessment is viable.

3. Estimating the public health impact of ambient air pollution using PM10 as indicator resulted in gains in life expectancy of approximately five months for males and females per decrease of 10 $\mu g/m^3$ PM_{10} in the base model which had effects only in the 30 years and above. Using a pollution reduction to 50 $\mu g/m^3$ PM_{10}, which is the current WHO guideline, the gains in life expectancy were approximately two years, more and less, for both males and females.

4. Incorporating the effects on infants and the elderly into the model resulted in bigger gains in life expectancy than the base model. Pollution reduction to 50 $\mu g/m^3$ PM_{10} showed that the gains in life expectancy compared to the base model could be as much as five months longer. Other pollution

reduction scenarios, namely using the national guideline of 60 $\mu g/m^3$ PM_{10} and background levels yielded gains in life expectancy proportional to the amount of reduction.

5. The US cohort studies had recently been re-analysed and found evidence of effect modification by educational level as a measure of socio-economic effects. Evidence from a time series study in Mexico City had similar findings. Exploring such effect modification among different educational levels in the Metropolitan Manila population found that people with low educational level had substantial gains in life expectancy as compared to those in the middle and high educational levels when exposure levels were reduced to 50 $\mu g/m^3$ PM_{10}.

6. Modifying the cardio-pulmonary causes of deaths alone in the life table analysis showed that the attributable deaths due to air pollution were dominated by the cardio-pulmonary causes. Hence the gains in life expectancy in modifying the cardio-pulmonary causes of death were substantial and comparable to the gains in life expectancy when total mortality was modified.

7. Other types of effect measures were explored such as percentage of those expected to survive to 65 and 75 years old for the 1995 birth cohort, and the reduction of premature deaths and life year gains within five years for the population alive in 1995 in both immediate and delayed effects scenarios. For the population alive in 1995, the scenarios showed that the reduction in the number of premature deaths within five years and years of life gained were dependent on the number of years the effects were delayed. More years were gained and premature deaths reduced in the immediate effect scenario or in scenarios with shorter delay of effects. For the 1995 birth cohort, apart from increases in gains in life expectancy in the three pollution reduction scenarios, the percentages of people expected to survive to 65 and 75 years old were also slightly increased compared with no pollution reduction scenario.

8. The predicted gains in life expectancies from a pollution reduction scenario vary markedly between cities, and there is evidence that the gains were higher in cities with lower educational levels.

9. The estimates that resulted from this assessment were found to be sensitive to the following: exposure-response coefficients, the exposure level, measures of heterogeneity, baseline mortality rates and time period of effect.

10. In estimating the public health impact of ambient air pollution using PM_{10} as indicator, a base model would yield conservative estimates. On the other hand, a model, which takes into account heterogeneity such as age and socio-economic status, would yield estimates that would reflect the present evidence seen in air pollution epidemiological studies. In addition, because this latter model would show effects in vulnerable groups, it may help to reinforce the urgency by which control measures are formulated and enforced. However, it must be noted that with more complex models, there would also be more assumptions than the more prudent model. Thus it is important that in dealing with complex models, these assumptions are assessed thoroughly and justified.

In addition to these conclusions, other implications need to be expressed including a recommendation for future studies in Metropolitan Manila on air pollution. These are the following:

- It is important that risks are communicated to the policy makers in a manner that would easily be understood. In considering the type of effect measure to be used and presented, it is vital that the concept of that effect measure is clear and well defined. However, even with such clear concepts of the effect measures, certain effect measures may still be more compelling than others. In this latter case, the element of outrage or the scare factor could play a role.

- Finally, there is a need to look in to the possibility of directly measuring the effects of air pollution on mortality to validate the findings in this assessment. It is quite difficult to conduct a prospective cohort study in Metropolitan Manila as this would be resource intensive and would take a long time, although such cohort studies are definitely needed. However, a retrospective cohort maybe possible. There were surveys done in the 1980's from which a cohort may be identified. The possibility of this endeavour naturally relies on whether the data sets for the surveys are still intact and available. In addition to the retrospective cohort design and with the monitoring stations to be improved in the next two years, there is also an option to conduct a time series study. A time series study would not measure long-term effects but it would at least give an indication of the exposure-response relationship of particulate with mortality in the local setting. In turn, this exposure-response coefficient could be used to adjust more accurately the cohort studies' coefficients for further assessments.

Afterword: Policy-makers and the Results

The environmental health risk assessment framework used in this study and its results were separately presented to different officials of the Department of Health (DOH) and the Department of Environment and Natural Resources-Environmental Management Bureau (DENR-EMB) either as a group and/or individually. The presentations were undertaken to help gain insight into the acceptability and intelligibility of alternative indicators among key implementing institutions.

The research results were individually explained to four medical practitioners within the DOH, including the Health Minister himself, two engineers and an environmental health consultant, who were all connected with the Department at the time the study was being undertaken. Likewise, a similar presentation was made to the Assistant Administrator of the Air Pollution Division of the DENR-EMB, which included five members of staff. This audience was asked what their views are on the policy implications of the results of this study and which effect measures tackled by the research do they find most compelling. There was a mixture of reactions, especially on the types of effect measures presented, among policy makers at different levels of management and in institutions with different foci.

Both the DENR and the DOH saw the importance of the results and the model, especially in reinforcing the Clean Air Act agenda. The Health Minister shared this view although he immediately attached a caveat, saying that "...the problem is that most control measures are not within the jurisdiction of the Department of Health." Recognition of the role of government institutions and specific offices within these institutions, or how they could participate in the formulation of control measures is quite often overlooked. The problem is usually readily recognised but the responsible unit and/or individual would almost always hesitate to act.

The Chief of the Air pollution Division of the DENR-EMB recognised that the results presented can be very important in the next round of evaluation of air quality standards. He put it succinctly with the following statement;

> *"Perhaps later when we review the standards, we can make use of these results and the technique by which these results were arrived at. The methodology would be very useful."*

Other members of staff of the air pollution division concurred with this appraisal[1].

There seems to be greater enthusiasm about the research on the part of the DOH than with their DENR-EMB counterpart. The reaction of DENR-EMB may partly be explained by, one, the lack of appreciation of the effect measures presented perhaps because of the paucity of their knowledge of these measures and, two, and perhaps more importantly, because of the political dynamic between the DOH and DENR, which is not exactly constructive.

Feedback on the effect measures was also gathered. The effect measures presented to the officials were in the form of life shortening, number of deaths avoided and life years gained if control measures were to be implemented. Immediate and delayed effect scenarios were also presented. The interviewees were asked which of these measures is the most compelling to drive them to action and/or to communicate the risk with the community. Both the Health Minister and the Policy Chief responded that life shortening was the most compelling. According to the health minister,

> *"...this effect measure would be most effective in advocacy in the community kasi personal (since it is more personal), people could easily relate to it and the scare factor is heightened."*

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[1] Such concurrence, however, can only be expected since it is customary among members of the national and local government agencies not to be very vocal about their actual opinion on such matters.

The policy chief in almost similar words expressed the same sentiment.

However, the other DOH officials chose 'number of deaths' as a more effective measure since, they believe, it to be the most easily understood. The environmental health chief believed that for policymakers to take action the number of deaths is the easiest to appreciate and, therefore, the most effective. She described the use of life expectancy as "...difficult to comprehend" compared to the number of deaths. Likewise, the Environmental Health Medical Officer shared the same view:

> *"... the number of deaths avoided since this is the most understandable and with large numbers, the scariest to look at."*

Thus for all four interviewees, the scare or outrage factor was the critical factor (169) either for the public or for the policymakers.

None of the DOH officials chose the life years gained as compelling. The consultant for the environmental and occupational division, who was also active in the academe, pointed out quite candidly, that using the life years gained together

> *"... with the economics of it will be influential (be)cause the policy makers are fond of equating everything with how much it would cost, including life."*

However, she added that

> *"...of course, in the end, the decisions made by them (i.e. politicians and political appointees) are mostly if not entirely political in nature."*

It is quite interesting to note the frank cynicism of an academic who has been working with government for quite sometime.

Other DOH officials feel that the life years gained and life shortening measures are too vague a concept especially for politicians. The EMB personnel, on the other hand, did not register any reaction on the matter. This may, perhaps,

be due to their limited understanding of the measures used, except perhaps for the number of deaths avoided. Although there were only a few individuals who were interviewed, the results of the exercise underline the importance of risk communication in the choices made by policymakers and scientists alike.

In analysing the different reactions of policymakers to the problem of air pollution, it would be prudent to explore its denotation in terms of a theoretical framework of risk analysis by decision makers. It is not the desire of this study to immerse deeply into the discussion of risk perception and communication but only to describe the responses of the governmental officials according to certain elements in risk analysis. In this regard, one helpful schema that is available on how policy makers might consider risk would be the Risk Baseball Diamond by Taylor. Taylor proposed that policy makers recognise and analyse the risk presented to them in four ways. These are the following:

- Data Problem
- Communication Problem
- Structural Problem
- Risk Culture Problem

First, the policymaker may categorise the risk as a data problem, nothing is conclusive and, therefore, an improved risk assessment must be undertaken. Second, the risk is a communication problem. Explaining the concepts of risk and risk measures would be needed so it can be completely understood. The public's perception of risk must likewise be carefully studied. Policy decisions would depend on the clarity of the public's understanding of the empirical data. Hence, it seems that these first two ways give more weight to the empirical evidence with the public perception to a lesser degree.

Third, policymakers see the problem as structural, which purports that public participation and openness are needed in decision making. They recognise that they cannot depend on science and technical experts all the time. Finally, policy makers analyse risk as a risk culture problem. Risk is perceived beyond the data presented. It is a socio-cultural issue that transcends science. This point recognizes that certain issues, development or technology will always have an

inherent risk or scientific uncertainty, and that bigger societal values must play a role in decision-making. In addition, Taylor points out how policymakers must take responsibility for their ultimate decision and be capable to defend it. Taylor originally conceived this framework to illustrate how the policy makers are faring with regards to the debate on the sale of genetically-modified foods (*Taylor, 1999*).

This risk baseball diamond framework only applies to the policy makers interviewed here to a certain extent. Some, if not most, of the interviewees are between bases. Unlike in the GM food debate, with air pollution regulation since it has been in existence for a while, it is more difficult to categorise the policy makers into the ways mentioned above. A slightly different interpretation would probably be more appropriate.

For example, most of those interviewed recognised that particulate pollution is indeed a problem and the pollution data, based on guidelines that have been set, says that it is. In addition, a number of local studies regarding particulate pollution on specific populations, e.g. jeepney drivers, school children, street children are available. However, the full extent to which it is a problem locally, i.e. public health impact on the whole Metropolitan Manila population, is not quite clear or appreciated. This lack of data or information may thus affect some decision-making. It did so in the case of formulating the national guidelines, by adapting the US standards, which may or may not be realistic and from the experience of recent years, not attainable. A data problem exists but not in terms of proving that air pollution is detrimental but how detrimental it is or has been for the local population. The reaction of the DENR-EMB personnel could be classified in the first way.

However, the DENR-EMB personnel can also be classified in the second way, a communication problem, since the extent of their understanding the quantitative risk assessment results presented them was not complete, probably because of the effect measures used. Nevertheless, the method of risk assessment was appreciated and deemed to be important for later use.

This communication problem could likewise become relevant when presented to the public. In this regard, the health minister and the policy chief

appreciated the significance of the life shortening effect measure. They have recognised that public participation could only occur if the way the public understand the consequences of particulate pollution could be personal enough such that public clamour would be inevitable. The outrage factor was clearly the motivation for such participation. (*Sandman, 1994*) The DOH personnel who were interviewed had the same opinion on the scare or outrage factor when they chose the most compelling effect measure. On the other hand, the health minister and the policy chief were of the view that the effect measure would affect risk communication to the general public. Consequently, the reaction of the two latter DOH officials could also be categorised within the third way since they hinted at openness to public participation and not being restricted by the data presented to them.

Eventually, these policy makers would have to make a decision that they would have to take responsibility for, one that is defensible. This is embodied in the last category of Taylor's framework. As to the extent to which those who were interviewed have taken decisions, it is difficult to conclude from the interviews alone.

Certain decisions have been made for the air pollution programme as discussed earlier (see DPSEEA framework - Clean Air Act etc). However, control and/ or preventative measures stemming from the full appreciation of the problem by the policy makers could still be instigated. The results of this study could help in this latter task.

As for the most compelling measures, it was unanimous that the most effective effect measure to react to an issue for policy makers is the measure that could easily be understood and with an element of outrage or scare factor. Furthermore, showing the economics of the issue could be a powerful tool for winning support.

Whereas those interviewed showed in part some concurrence with Taylor's framework, the reactions and responses were by no means completely compatible.

This section had described the reactions of some officials from two government agencies involved in environmental health, on the results of this risk assessment. The reactions brought some quite interesting insights that can be useful in risk communication among decision-makers. An attempt to define these reactions in the context of a framework was likewise undertaken.

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Annexes

Annex 1: Females, Metropolitan Manila, 1995

Age Grp	Population	Cases	Rate	N	nQ_x	nP_x	l_x	nd_x	L_x	T_x	E_x
Under 1	129201	2548	0.0197	1	0.019567	0.980433	100000	1956.686	98825.99	7177581	71.77581
1 to 4	455632	877	0.0019	4	0.007676	0.992324	98043.31	752.5371	390367.2	7078755	72.20029
5 to 9	478433	308	0.0006	5	0.003214	0.996786	97290.78	312.6604	485672.2	6688388	68.74638
10 to 14	433907	261	0.0006	5	0.003003	0.996997	96978.12	291.2293	484162.5	6202716	63.95996
15 to 19	556091	304	0.0005	5	0.00273	0.99727	96686.89	263.9199	482774.6	5718554	59.14508
20 to 24	581667	430	0.0007	5	0.003689	0.996311	96422.97	355.7481	481225.5	5235779	54.30012
25 to 29	523779	510	0.0010	5	0.004857	0.995143	96067.22	466.5642	479169.7	4754553	49.49194
30 to 34	412827	620	0.0015	5	0.007481	0.992519	95600.66	715.199	476215.3	4275384	44.72128
35 to 39	340462	644	0.0019	5	0.009413	0.990587	94885.46	893.178	472194.3	3799168	40.03952
40 to 44	260845	758	0.0029	5	0.014425	0.985575	93992.28	1355.83	466571.8	3326974	35.39625
45 to 49	198000	860	0.0043	5	0.021484	0.978516	92636.45	1990.191	458206.8	2860402	30.87772
50 to 54	134158	898	0.0067	5	0.032917	0.967083	90646.26	2983.818	445771.7	2402196	26.50077
55 to 59	109144	1074	0.0098	5	0.04802	0.95198	87662.44	4209.528	427788.4	1956424	22.3177
60 to 64	82439	1297	0.0157	5	0.075687	0.924313	83452.91	6316.324	401473.7	1528635	18.31734
65 to 69	57067	1440	0.0252	5	0.118681	0.881319	77136.59	9154.622	362796.4	1127162	14.61254
70 to 74	37251	1545	0.0415	5	0.187894	0.812106	67981.97	12773.44	307976.2	764365.3	11.24365
75 to 79	22758	1588	0.0698	5	0.297067	0.702933	55208.53	16400.62	235041.1	456389	8.26664
80 to 84	14217	1812	0.1275	5	0.483277	0.516723	38807.91	18754.98	147152.1	221347.9	5.703681
85 and up	8605	2108	0.2450	3.7	0.623728	0.376272	20052.93	12507.58	74195.83	74195.83	3.7
all ages	4836483	19882	0.0041								

Annex 2: Metropolitan Manila Females, 1995 using 3 relative risks for excess of 35.6 µmg/m^3 PM_{10}

Age Grp	Population	Cases	Rate	N	RR	Clean Air ASDR	nQx	nPx	nlx	ndx	Lx	Tx	Ex
Under 1	129,201	2,548	0.02	1.00	1.15	0.02	0.02	0.98	100,000.00	1,703.20	98,978.08	7,344,635.28	73.45
1 to 4	455,632	877	0.00	4.00	1.00	0.00	0.01	0.99	98,296.80	754.48	391,376.42	7,245,657.20	73.71
5 to 9	478,433	308	0.00	5.00	1.00	0.00	0.00	1.00	97,542.31	313.47	486,927.89	6,854,280.78	70.27
10 to 14	433,907	261	0.00	5.00	1.00	0.00	0.00	1.00	97,228.84	291.98	485,414.26	6,367,352.88	65.49
15 to 19	556,091	304	0.00	5.00	1.00	0.00	0.00	1.00	96,936.86	264.60	484,022.80	5,881,938.62	60.68
20 to 24	581,667	430	0.00	5.00	1.00	0.00	0.00	1.00	96,672.26	356.67	482,469.63	5,397,915.82	55.84
25 to 29	523,779	510	0.00	5.00	1.00	0.00	0.00	1.00	96,315.59	467.77	480,408.53	4,915,446.19	51.03
30 to 34	412,827	620	0.00	5.00	1.18	0.00	0.01	0.99	95,847.82	608.01	477,719.07	4,435,037.66	46.27
35 to 39	340,462	644	0.00	5.00	1.18	0.00	0.01	0.99	95,239.81	760.30	474,298.27	3,957,318.59	41.55
40 to 44	260,845	758	0.00	5.00	1.18	0.00	0.01	0.99	94,479.50	1,156.24	469,506.92	3,483,020.32	36.87
45 to 49	198,000	860	0.00	5.00	1.18	0.00	0.02	0.98	93,323.27	1,701.90	462,361.59	3,013,513.39	32.29
50 to 54	134,158	898	0.01	5.00	1.18	0.01	0.03	0.97	91,621.37	2,562.29	451,701.12	2,551,151.80	27.84
55 to 59	109,144	1,074	0.01	5.00	1.18	0.01	0.04	0.96	89,059.08	3,637.55	436,201.50	2,099,450.68	23.57
60 to 64	82,439	1,297	0.02	5.00	1.18	0.01	0.06	0.94	85,421.52	5,510.90	413,330.36	1,663,249.18	19.47
65 to 69	57,067	1,440	0.03	5.00	1.20	0.02	0.10	0.90	79,910.62	7,982.15	379,597.74	1,249,918.82	15.64
70 to 74	37,251	1,545	0.04	5.00	1.20	0.03	0.16	0.84	71,928.47	11,441.62	331,038.31	870,321.08	12.10
75 to 79	22,758	1,588	0.07	5.00	1 20	0.06	0.25	0.75	60,486.85	15,353.96	264,049.37	539,282.77	8.92
80 to 84	14,217	1,812	0.13	5.00	1.20	0.11	0.42	0.58	45,132.90	18,939.16	178,316.58	275,233.40	6.10
85 and up	8,605	2,108	0.24	3.70	1.20	0.20	0.55	0.45	26,193.74	14,361.28	96,916.82	96,916.82	3.70
all ages	4,836,483	9,909	0.00										

Annex 2: AIR POLLUTION POLICY FRAMEWORK

Using the DPSEEA framework and Environmental Health as Focus for Metropolitan Manila

STAGE	PROCESS	ACTIVITY	ACTION/POLICY INSTRUMENT	COMMENTS/IMPLEMENTATION ISSUES
DRIVING FORCE	Type of Development or Human Activities	Economic development leading to increased number of industries & transport vehicles	Legislation regarding the coming in of industries, mass transport system and transport vehicle manufacture and importation. Clean Air Act	The Clean Air Act has been legislated and implementing guidelines have been formulated. Implementation was supposed to have started at the end of November, 2000. However, due to the political situation and some questions on the guidelines, this has been postponed. Also, an Air Pollution Abatement Programme for Metropolitan Manila that includes the mass transport system has been proposed concomitantly.
PRESSURE	Source Activities	1.Fuel consumption – quality and quantity 2.Road network activities – quantity and quality 3.Equipment and Transport types – quantity and quality	1.Fuel Quality Standards – limit lead and sulphur content, volatility limits and cleaner octane booster 2.Mass Transport systems will limit fuel consumption 3.Improve and increase road networks with regular maintenance programme 4.Traffic Management 5.Promote cleaner engines and equipment ; early scrappage incentives 6.Compulsory Fitting of Pollution control devices 7.Cost-benefit analysis of the activities mentioned here vs. the cost of pollution including losses because of health problems	1. Fuel quality has been improving in the past 5 years with the adoption of the low-lead and unleaded gasoline for motor vehicles. Also sulphur content is being reduced this year. Other aspects of fuel quality are at moment under study. 2. Mass transport system has been operational in the three major thoroughfares of Metro Manila. More light rail transit systems are being planned in other parts of the metropolis. These trains have decreased considerably the use of buses and jeepneys in the said roads. However, due to the confined spaces underneath the train systems where the other vehicles pass, the pollutants are quite concentrated. 3. Road improvement and increasing the network is being done. However, the increase and improvement, can not cope with the increase in the number of motor vehicles every year on the road. 4. Traffic management due to the sheer volume of vehicles and the amount of roads including some questionable driving habits and lack of implementation of the traffic laws, is very difficult. Several schemes are operational, e.g., colour coding wherein the last number on your plate will determine whether you could use your car that day or not, yellow lane for buses etc. Although,

				the schemes have decreased the amount of vehicles slightly, they do not work completely. 5. Early scrappage is controversial and is perceived as anti-poor. Cleaner engines and equipment for industries and vehicles alike are being promoted by the government at present. 6. Pollution control devices in industries are compulsory. Not having such for machines and equipment would mean fines. However, the fines are not very restrictive so that industries sometimes would rather pay the fine every month rather than buy the control devices which are very expensive. In the Clean Air Act, this is being addressed.
	Emissions	Increased source emissions of criteria pollutants, e.g. PM, SO_2 etc & other pollutants such as benzene and lead	1.Mandated Emissions Standards 2.Inspection and maintenance schemes; roadside inspection programme	1. The emissions standards are mandated and contained in the Clean Air Act and due for review every two years or as the need arises. 2. Inspection and maintenance schemes including roadside inspections are done on a piecemeal basis. Inspection before issuing a license is made by the Land Transportation Office but has been criticised vehemently because of corrupt practices.
STATE	**Environmental Levels**	Deteriorating Air Quality. Increased airborne criteria and non-criteria pollutants	1.Mandated Air Quality Standards 2.Monitoring Programme of air pollutants 3.Reporting of and regular review(feedback) system for air quality data	1. The air quality standards have been mandated and are contained in the Clean Air Act as the emission standards are as well. However, like the emission standards, these air quality standards are all adapted from foreign guidelines and have not been reviewed as to its applicability in the local setting. Its review will be done every two years. 2. As of now, most of the monitoring stations are non-functional and the network is in the process of being revived. The Clean Air Act has provided two years or by the year 2003, for the network to be fully functional. 3. The feedback system has not been clearly established. Even the Clean Air act has not addressed this issue in detail.
EXPOSURE	Human Exposure	Increased population in disadvantaged areas, i.e. living near the main	Land Zoning for industries and major roads	Land zoning has always been there but is perceived to be changing all the time depending on the needs of the moment. The industries are so entwined with the residents

		thoroughfares and around industrial complexes		that it is difficult at this point to strictly enforce the land zoning.
EFFECTS	Early/Moderate/ Advanced	Increase in cardiopulmonary cases and deaths (COPD, MIs, Cancer etc.) especially among the elderly and the susceptible groups including, as per findings in this study, the low socio-economic classes Increase in road accidents Long term-effect on reduction in I.Q. points	1. A progressively constructed policy must be formulated to address the issues raised in this assessment. 2.Road Safety programme e.g. seatbelt implementation and education, clearer road signs and strict implementation of traffic laws 3.Regular monitoring of blood lead levels 4.Research to validate the findings in this study: Studies which will investigate the exposure-response relationship within Metropolitan Manila.	1. There is no policy or programme as of the moment regarding cardio-respiratory health care for the susceptible. However, there are general programmes for the young especially on acute respiratory illnesses, also on family health and infectious diseases and some other programmes not related to air pollution. No programme that I know of exists for the elderly. 2. There is an ongoing road safety programme in the metropolitan especially regarding the use of seatbelts. However, other aspects of the programme have been lacking i.e. continuous traffic education of drivers and commuters. 3.No regular monitoring of blood lead has occurred. However, the Department of Health has undertaken a system wherein blood lead could be measured every other year for the next six years among school children. The latest blood lead measurements yielded similar results as that done in 1993 before the introduction of low-lead and unleaded gasoline. Other sources of lead are being investigated. 4. These studies are, at present , being proposed to the WHO and ADB for funding by the Department of Health. Its implementation is expected to begin early next year.

Annex 3: Life Tables

Life Expectancy Tables 1:Comparison of Pollution Scenarios using only 1 relative risk coefficient - 1.05- for 30 years and above

for Metropolitan Manila, 1995

1.1. Reduction to 60 ug/m3

NCR (Male)	Clean Air Computations					Clean Air ASDR	nQx	nPx	nlx	ndx	Lx	Tx	Ex
Age Grp	Population	Cases	Rate	N	RR								
Under 1	140,415	3634	0.0259	1	1	0.02588	0.025615	0.974385	100000	2561.525	98463.08	6583136	65.83136
1 to 4	487,282	1110	0.0023	4	1	0.002278	0.009079	0.990921	97438.47	884.6125	387630.8	6484673	66.55146
5 to 9	509576	421	0.0008	5	1	0.000826	0.004122	0.995878	96553.86	398.0308	481774.2	6097042	63.14654
10 to 14	438531	311	0.0007	5	1	0.000709	0.00354	0.99646	96155.83	340.3584	479928.3	5615268	58.39758
15 to 19	463906	570	0.0012	5	1	0.001229	0.006125	0.993875	95815.47	586.8384	477610.3	5135340	53.59614
20 to 24	496600	1180	0.0024	5	1	0.002376	0.011811	0.988189	95228.63	1124.71	473331.4	4657729	48.91102
25 to 29	487562	1414	0.0029	5	1	0.0029	0.014396	0.985604	94103.92	1354.752	467132.7	4184398	44.46571
30 to 34	397990	1380	0.0035	5	1.19	0.002914	0.014464	0.985536	92749.17	1341.491	460392.1	3717265	40.07869
35 to 39	337886	1458	0.0043	5	1.19	0.003626	0.017968	0.982032	91407.68	1642.381	452932.5	3256873	35.63019
40 to 44	260181	1539	0.0059	5	1.19	0.004971	0.024548	0.975452	89765.3	2203.591	443317.5	2803941	31.23635
45 to 49	200036	1854	0.0093	5	1.19	0.007789	0.038199	0.961801	87561.71	3344.751	429446.7	2360623	26.95954
50 to 54	129822	1763	0.0136	5	1.19	0.011412	0.055477	0.944523	84216.96	4672.075	409404.6	1931176	22.93097
55 to 59	98814	2074	0.0210	5	1.19	0.017638	0.084464	0.915536	79544.88	6718.709	380927.6	1521772	19.13098
60 to 64	70088	2204	0.0314	5	1.19	0.026425	0.123939	0.876061	72826.17	9026.002	341565.9	1140844	15.6653
65 to 69	45585	2172	0.0477	5	1.19	0.040057	0.182055	0.817945	63800.17	11615.13	289963	799278.2	12.52784
70 to 74	26416	1951	0.0739	5	1.19	0.062064	0.26864	0.73136	52185.04	14018.99	225877.7	509315.2	9.759793
75 to 79	14303	1594	0.1114	5	1.19	0.093651	0.379423	0.620577	38166.05	14481.08	154627.6	283437.5	7.426428
80 to 84	7664	1302	0.1699	5	1.19	0.142761	0.526054	0.473946	23684.97	12459.57	87275.92	128809.9	5.438466
85 and up	4,920	1165	0.2368	3.7	1.19	0.198982	0.538136	0.461864	11225.4	6040.797	41533.98	41533.98	3.7
all ages	4,617,557	29199	0.0063										

1.1. Females				Excess PM10 of 35.6ug/m3									
Age Grp	Population	Cases	Rate		RR	Clean Air ASDR	nQx	nPx	nlx	ndx	Lx	Tx	Ex
Under 1	129201	2548	0.0197	1	1	0.019721	0.019567	0.980433	100000	1956.686	98825.99	7325383	73.25383
1 to 4	455632	877	0.0019	4	1	0.001925	0.007676	0.992324	98043.31	752.5371	390367.2	7226557	73.7078
5 to 9	478433	308	0.0006	5	1	0.000644	0.003214	0.996786	97290.78	312.6604	485672.2	6836190	70.26555
10 to 14	433907	261	0.0006	5	1	0.000602	0.003003	0.996997	96978.12	291.2293	484162.5	6350517	65.48402
15 to 19	556091	304	0.0005	5	1	0.000547	0.00273	0.99727	96686.89	263.9199	482774.6	5866355	60.67374
20 to 24	581667	430	0.0007	5	1	0.000739	0.003689	0.996311	96422.97	355.7481	481225.5	5383580	55.83297
25 to 29	523779	510	0.0010	5	1	0.000974	0.004857	0.995143	96067.22	466.5642	479169.7	4902355	51.03046
30 to 34	412827	620	0.0015	5	1.19	0.001262	0.00629	0.99371	95600.66	601.3667	476499.9	4423185	46.26731
35 to 39	340462	644	0.0019	5	1.19	0.00159	0.007916	0.992084	94999.29	752.0353	473116.4	3946685	41.54437
40 to 44	260845	758	0.0029	5	1.19	0.002442	0.012136	0.987864	94247.25	1143.761	468376.9	3473569	36.85592
45 to 49	198000	860	0.0043	5	1.19	0.00365	0.018085	0.981915	93103.49	1683.749	461308.1	3005192	32.27797
50 to 54	134158	898	0.0067	5	1.19	0.005625	0.027734	0.972266	91419.74	2535.468	450760	2543884	27.82642
55 to 59	109144	1074	0.0098	5	1.19	0.008269	0.040508	0.959492	88884.28	3600.525	435420.1	2093124	23.54887
60 to 64	82439	1297	0.0157	5	1.19	0.013221	0.063989	0.936011	85283.75	5457.256	412775.6	1657704	19.43751
65 to 69	57067	1440	0.0252	5	1.19	0.021205	0.100686	0.899314	79826.49	8037.378	379039	1244928	15.59543
70 to 74	37251	1545	0.0415	5	1.19	0.034853	0.160299	0.839701	71789.12	11507.72	330176.3	865889.2	12.06157
75 to 79	22758	1588	0.0698	5	1.19	0.058637	0.2557	0.7443	60281.39	15413.95	262872.1	535712.9	8.88687
80 to 84	14217	1812	0.1275	5	1.19	0.107103	0.422412	0.577588	44867.44	18952.57	176955.8	272840.8	6.081043
85 and up	8605	2108	0.2450	3.7	1.19	0.20586	0.551608	0.448392	25914.87	14294.85	95885.04	95885.04	3.7
all ages	4836483	19909	0.0041										

1.2. Reduction to background level

NCR (Male)	Clean Air Computations	Pooled estimate		Excess of 66.35 ug/m3PM10									
Age Grp	Population	Cases	Rate	N	RR	Clean Air A	nQx	nPx	nlx	ndx	Lx	Tx	Ex
Under 1													
1 to 4	140,415	3634	0.0259	1	1	0.02588	0.025615	0.974385	100000	2561.525	98463.08	6728806	67.28806
5 to 9	487,282	1110	0.0023	4	1	0.002278	0.009079	0.990921	97438.47	884.6125	387630.8	6630343	68.04646
10 to 14	509576	421	0.0008	5	1	0.000826	0.004122	0.995878	96553.86	398.0308	481774.2	6242713	64.65523
15 to 19	438531	311	0.0007	5	1	0.000709	0.00354	0.99646	96155.83	340.3584	479928.3	5760938	59.91252
20 to 24	463906	570	0.0012	5	1	0.001229	0.006125	0.993875	95815.47	586.8384	477610.3	5281010	55.11646
25 to 29	496600	1180	0.0024	5	1	0.002376	0.011811	0.988189	95228.63	1124.71	473331.4	4803400	50.44071
30 to 34	487562	1414	0.0029	5	1	0.0029	0.014396	0.985604	94103.92	1354.752	467132.7	4330068	46.01369
35 to 39	397990	1380	0.0035	5	1.38	0.002513	0.012485	0.987515	92749.17	1157.946	460851	3862936	41.64927
40 to 44	337886	1458	0.0043	5	1.38	0.003127	0.015513	0.984487	91591.23	1420.857	454404	3402085	37.14422
45 to 49	260181	1539	0.0059	5	1.38	0.004286	0.021204	0.978796	90170.37	1912.004	446071.8	2947681	32.69013
50 to 54	200036	1854	0.0093	5	1.38	0.006716	0.033026	0.966974	88258.37	2914.855	434004.7	2501609	28.34416
55 to 59	129822	1763	0.0136	5	1.38	0.009841	0.048022	0.951978	85343.51	4098.363	416471.6	2067604	24.22685
60 to 64	98814	2074	0.0210	5	1.38	0.015209	0.073261	0.926739	81245.15	5952.117	391345.4	1651132	20.32284
65 to 69	70088	2204	0.0314	5	1.38	0.022787	0.107795	0.892205	75293.03	8116.184	356174.7	1259787	16.73179
70 to 74	45565	2172	0.0477	5	1.38	0.034542	0.158982	0.841018	67176.85	10679.9	309184.5	903612.3	13.45125
75 to 79	26416	1951	0.0739	5	1.38	0.053519	0.236018	0.763982	56496.95	13334.3	249149	594427.8	10.52141
80 to 84	14303	1594	0.1114	5	1.38	0.080757	0.335959	0.664041	43162.65	14500.88	179561.1	345278.8	7.999482
85 and up	7664	1302	0.1699	5	1.38	0.123105	0.470671	0.529329	28661.77	13490.26	109583.2	165717.8	5.78184
all ages	4,920	1165	0.2368	3.7	1.38	0.171586	0.481897	0.518103	15171.51	7311.111	56134.59	56134.59	3.7
	4,617,557	29199	0.0063										

1.2. Females				Excess PM10 of 66.35 ug/m3									
Age Grp	Population	Cases	Rate		RR	Clean Air A	nQx	nPx	nlx	ndx	Lx	Tx	Ex
Under 1	129201	2548	0.0197	1	1	0.019721	0.019567	0.980433	100000	1956.686	98825.99	7443882	74.43882
1 to 4	455632	877	0.0019	4	1	0.001925	0.007676	0.992324	98043.31	752.5371	390367.2	7345056	74.91644
5 to 9	478433	308	0.0006	5	1	0.000644	0.003214	0.996786	97290.78	312.6604	485672.2	6954689	71.48353
10 to 14	433907	261	0.0006	5	1	0.000602	0.003003	0.996997	96978.12	291.2293	484162.5	6469016	66.70594
15 to 19	556091	304	0.0005	5	1	0.000547	0.00273	0.99727	96686.89	263.9199	482774.6	5984854	61.89933
20 to 24	581667	430	0.0007	5	1	0.000739	0.003689	0.996311	96422.97	355.7481	481225.5	5502079	57.06192
25 to 29	523779	510	0.0010	5	1	0.000974	0.004857	0.995143	96067.22	466.5642	479169.7	5020854	52.26396
30 to 34	412827	620	0.0015	5	1.38	0.001088	0.005427	0.994573	95600.66	518.7945	476706.3	4541684	47.50683
35 to 39	340462	644	0.0019	5	1.38	0.001371	0.00683	0.99317	95081.86	649.4118	473785.8	4064978	42.7524
40 to 44	260845	758	0.0029	5	1.38	0.002106	0.010474	0.989526	94432.45	989.0507	469689.6	3591192	38.02922
45 to 49	198000	860	0.0043	5	1.38	0.003147	0.015614	0.984386	93443.4	1459.046	463569.4	3121502	33.40527
50 to 54	134158	898	0.0067	5	1.38	0.00485	0.023962	0.976038	91984.35	2204.094	454411.5	2657933	28.89549
55 to 59	109144	1074	0.0098	5	1.38	0.007131	0.035029	0.964971	89780.26	3144.868	441039.1	2203521	24.5435
60 to 64	82439	1297	0.0157	5	1.38	0.011401	0.055423	0.944577	86635.39	4801.629	421172.9	1762482	20.34368
65 to 69	57067	1440	0.0252	5	1.38	0.018285	0.087429	0.912571	81833.76	7154.651	391282.2	1341309	16.39066
70 to 74	37251	1545	0.0415	5	1.38	0.030055	0.139771	0.860229	74679.11	10437.99	347300.6	950027.3	12.72146
75 to 79	22758	1588	0.0698	5	1.38	0.050564	0.224446	0.775554	64241.12	14418.64	285159	602726.7	9.382257
80 to 84	14217	1812	0.1275	5	1.38	0.092357	0.375164	0.624836	49822.48	18691.58	202383.4	317567.7	6.373985
85 and up	8605	2108	0.2450	3.7	1.38	0.177517	0.494437	0.505563	31130.89	15392.27	115184.3	115184.3	3.7
all ages	4836483	19909	0.0041										

1.3. Reduction to 50 ug/m3													
NCR (Male)	Clean Air Computations	Pooled estimate		Excess of 45.6 ug/m3PM10									
Age Grp	Population	Cases	Rate	N	RR	Clean Air A	nQx	nPx	nlx	ndx	Lx	Tx	Ex
Under 1													
1 to 4	140,415	3634	0.0259	1	1	0.02588	0.025615	0.974385	100000	2561.525	98463.08	6631928	66.31928
5 to 9	487,282	1110	0.0023	4	1	0.002278	0.009079	0.990921	97438.47	884.6125	387630.8	6533465	67.05221
10 to 14	509576	421	0.0008	5	1	0.000826	0.004122	0.995878	96553.86	398.0308	481774.2	6145834	63.65187
15 to 19	438531	311	0.0007	5	1	0.000709	0.00354	0.99646	96155.83	340.3584	479928.3	5664060	58.90501
20 to 24	463906	570	0.0012	5	1	0.001229	0.006125	0.993875	95815.47	586.8384	477610.3	5184132	54.10537
25 to 29	496600	1180	0.0024	5	1	0.002376	0.011811	0.988189	95228.63	1124.71	473331.4	4706521	49.42338
30 to 34	487562	1414	0.0029	5	1	0.0029	0.014396	0.985604	94103.92	1354.752	467132.7	4233190	44.9842
35 to 39	397990	1380	0.0035	5	1.25	0.002774	0.013774	0.986226	92749.17	1277.543	460552	3766057	40.60475
40 to 44	337886	1458	0.0043	5	1.25	0.003452	0.017113	0.982887	91471.63	1565.315	453444.9	3305505	36.13694
45 to 49	260181	1539	0.0059	5	1.25	0.004732	0.023384	0.976616	89906.31	2102.353	444275.7	2852060	31.72258
50 to 54	200036	1854	0.0093	5	1.25	0.007415	0.036399	0.963601	87803.96	3195.943	431029.9	2407785	27.42228
55 to 59	129822	1763	0.0136	5	1.25	0.010864	0.052884	0.947116	84608.02	4474.426	411854	1976755	23.36368
60 to 64	98814	2074	0.0210	5	1.25	0.016791	0.080573	0.919427	80133.59	6456.637	384526.4	1564901	19.52865
65 to 69	70088	2204	0.0314	5	1.25	0.025157	0.118342	0.881658	73676.96	8719.073	346587.1	1180374	16.02094
70 to 74	45565	2172	0.0477	5	1.25	0.038135	0.174077	0.825923	64957.88	11307.66	296520.3	833787.2	12.83581
75 to 79	26416	1951	0.0739	5	1.25	0.059085	0.257405	0.742595	53650.22	13809.83	233726.5	537266.9	10.01425
80 to 84	14303	1594	0.1114	5	1.25	0.089156	0.36453	0.63547	39840.39	14523.03	162894.4	303540.4	7.61891
85 and up	7664	1302	0.1699	5	1.25	0.135908	0.507207	0.492793	25317.36	12841.14	94483.96	140646	5.555317
all ages	4,920	1165	0.2368	3.7	1.25	0.189431	0.519009	0.480991	12476.22	6475.273	46162.02	46162.02	3.7

1.3.Females				Excess PM10 of 45.6 ug/m3									
Age Grp	Population	Cases	Rate		RR	Clean Air A	nQx	nPx	nlx	ndx	Lx	Tx	Ex
..													
..													
Under 1	129201	2548	0.0197	1	1	0.019721	0.019567	0.980433	100000	1956.686	98825.99	7365527	73.65527
1 to 4	455632	877	0.0019	4	1	0.001925	0.007676	0.992324	98043.31	752.5371	390367.2	7266701	74.11725
5 to 9	478433	308	0.0006	5	1	0.000644	0.003214	0.996786	97290.78	312.6604	485672.2	6876334	70.67816
10 to 14	433907	261	0.0006	5	1	0.000602	0.003003	0.996997	96978.12	291.2293	484162.5	6390661	65.89797
15 to 19	556091	304	0.0005	5	1	0.000547	0.00273	0.99727	96686.89	263.9199	482774.6	5906499	61.08893
20 to 24	581667	430	0.0007	5	1	0.000739	0.003689	0.996311	96422.97	355.7481	481225.5	5423724	56.2493
25 to 29	523779	510	0.0010	5	1	0.000974	0.004857	0.995143	96067.22	466.5642	479169.7	4942499	51.44834
30 to 34	412827	620	0.0015	5	1.25	0.001201	0.005989	0.994011	95600.66	572.5876	476571.8	4463329	46.68722
35 to 39	340462	644	0.0019	5	1.25	0.001513	0.007538	0.992462	95028.07	716.2906	473349.6	3986757	41.95347
40 to 44	260845	758	0.0029	5	1.25	0.002325	0.011557	0.988443	94311.78	1089.923	468834.1	3513408	37.25312
45 to 49	198000	860	0.0043	5	1.25	0.003475	0.017224	0.982776	93221.85	1605.664	462095.1	3044574	32.65944
50 to 54	134158	898	0.0067	5	1.25	0.005355	0.026421	0.973579	91616.19	2420.564	452029.5	2582478	28.18801
55 to 59	109144	1074	0.0098	5	1.25	0.007872	0.038601	0.961399	89195.63	3443.054	437370.5	2130449	23.88513
60 to 64	82439	1297	0.0157	5	1.25	0.012586	0.061012	0.938988	85752.57	5231.902	415683.1	1693078	19.74376
65 to 69	57067	1440	0.0252	5	1.25	0.020187	0.096085	0.903915	80520.67	7736.818	383261.3	1277395	15.86419
70 to 74	37251	1545	0.0415	5	1.25	0.03318	0.153194	0.846806	72783.85	11150.05	336044.1	894134	12.28479
75 to 79	22758	1588	0.0698	5	1.25	0.055822	0.244929	0.755071	61633.8	15095.93	270429.2	558089.9	9.054932
80 to 84	14217	1812	0.1275	5	1.25	0.101962	0.406255	0.593745	46537.87	18906.25	185423.7	287660.7	6.181217
85 and up	8605	2108	0.2450	3.7	1.25	0.195979	0.532176	0.467824	27631.61	14704.89	102237	102237	3.7
all ages	4836483	19909	0.0041										

Life Expectancy Tables 2 :Comparison of Pollution Scenarios using three different relative risk coefficients													
for corresponding age groups (1.04 - infants, 1.048- 30-64 y/o, 1.052- 65 y/o and above) for Metropolitan Manila, 1995													
2.1 Reduction to 60 ug/m3													
NCR (Male)	Clean Air Computations	Pooled estimate				Clean Air ASDR	nQx	nPx	nlx	ndx	Lx	Tx	Ex
Age Grp	Population	Cases	Rate	N	RR								
Under 1	140,415	3634	0.0259	1	1.15	0.0225	0.0223	0.9777	100000	2230.39	98661.76	6603794	66.03794
1 to 4	487,282	1110	0.0023	4	1	0.0023	0.0091	0.9909	97769.61	887.62	388948.1	6505133	66.53533
5 to 9	509576	421	0.0008	5	1	0.0008	0.0041	0.9959	96881.99	399.38	483411.5	6116185	63.13028
10 to 14	438531	311	0.0007	5	1	0.0007	0.0035	0.9965	96482.6	341.52	481559.2	5632773	58.38123
15 to 19	463906	570	0.0012	5	1	0.0012	0.0061	0.9939	96141.09	588.83	479233.4	5151214	53.57973
20 to 24	496600	1180	0.0024	5	1	0.0024	0.0118	0.9882	95552.26	1128.53	474939.9	4671981	48.89451
25 to 29	487562	1414	0.0029	5	1	0.0029	0.0144	0.9856	94423.72	1359.36	468720.2	4197041	44.44901
30 to 34	397990	1380	0.0035	5	1.18	0.0029	0.0146	0.9854	93064.37	1357.37	461928.4	3728320	40.06174
35 to 39	337886	1458	0.0043	5	1.18	0.0037	0.0181	0.9819	91706.99	1661.60	454381	3266392	35.6177
40 to 44	260181	1539	0.0059	5	1.18	0.0050	0.0248	0.9752	90045.4	2228.97	444654.6	2812011	31.22881
45 to 49	200036	1854	0.0093	5	1.18	0.0079	0.0385	0.9615	87816.43	3382.36	430626.2	2367356	26.95801
50 to 54	129822	1763	0.0136	5	1.18	0.0115	0.0559	0.9441	84434.07	4722.70	410363.6	1936730	22.93778
55 to 59	98814	2074	0.0210	5	1.18	0.0178	0.0851	0.9149	79711.36	6787.40	381588.3	1526367	19.14867
60 to 64	70088	2204	0.0314	5	1.18	0.0266	0.1249	0.8751	72923.96	9109.93	341845	1144778	15.69824
65 to 69	45585	2172	0.0477	5	1.2	0.0397	0.1807	0.8193	63814.03	11529.59	290246.2	802933.3	12.58239
70 to 74	26416	1951	0.0739	5	1.2	0.0615	0.2667	0.7333	52284.45	13944.25	226561.6	512687.1	9.805728
75 to 79	14303	1594	0.1114	5	1.2	0.0929	0.3769	0.6231	38340.19	14448.77	155579	286125.5	7.462807
80 to 84	7664	1302	0.1699	5	1.2	0.1416	0.5228	0.4772	23891.42	12490.82	88230.06	130546.4	5.464156
85 and up	4,920	1165	0.2368	3.7	1.2	0.1973	0.5349	0.4651	11400.6	6097.63	42182.23	42316.39	3.711768
all ages	4,617,557	29096	0.0063										

2.1. Females				Excess PM10 of 35.6ug/m3									
Age Grp	Population	Cases	Rate		RR	Clean Air ASDR	nQx	nPx	nlx	ndx	Lx	Tx	Ex
Under 1	129201	2548	0.0197	1	1.15	0.017149	0.017032	0.982968	100000	1703.205	98978.08	7344635	73.44635
1 to 4	455632	877	0.0019	4	1	0.001925	0.007676	0.992324	98296.8	754.4827	391376.4	7245657	73.71204
5 to 9	478433	308	0.0006	5	1	0.000644	0.003214	0.996786	97542.31	313.4687	486927.9	6854281	70.26982
10 to 14	433907	261	0.0006	5	1	0.000602	0.003003	0.996997	97228.84	291.9822	485414.3	6367353	65.48831
15 to 19	556091	304	0.0005	5	1	0.000547	0.00273	0.99727	96936.86	264.6023	484022.8	5881939	60.67804
20 to 24	581667	430	0.0007	5	1	0.000739	0.003689	0.996311	96672.26	356.6679	482469.6	5397916	55.83728
25 to 29	523779	510	0.0010	5	1	0.000974	0.004857	0.995143	96315.59	467.7705	480408.5	4915446	51.03479
30 to 34	412827	620	0.0015	5	1.18	0.001273	0.006344	0.993656	95847.82	608.0148	477719.1	4435038	46.27166
35 to 39	340462	644	0.0019	5	1.18	0.001603	0.007983	0.992017	95239.81	760.3031	474298.3	3957319	41.5511
40 to 44	260845	758	0.0029	5	1.18	0.002463	0.012238	0.987762	94479.5	1156.237	469506.9	3483020	36.86535
45 to 49	198000	860	0.0043	5	1.18	0.003681	0.018237	0.981763	93323.27	1701.896	462361.6	3013513	32.29113
50 to 54	134158	898	0.0067	5	1.18	0.005673	0.027966	0.972034	91621.37	2562.294	451701.1	2551152	27.84451
55 to 59	109144	1074	0.0098	5	1.18	0.008339	0.040844	0.959156	89059.08	3637.555	436201.5	2099451	23.57369
60 to 64	82439	1297	0.0157	5	1.18	0.013333	0.064514	0.935486	85421.52	5510.9	413330.4	1663249	19.47108
65 to 69	57067	1440	0.0252	5	1.2	0.021028	0.099888	0.900112	79910.62	7982.149	379597.7	1249919	15.64146
70 to 74	37251	1545	0.0415	5	1.2	0.034563	0.159069	0.840931	71928.47	11441.62	331038.3	870321.1	12.09981
75 to 79	22758	1588	0.0698	5	1.2	0.058148	0.25384	0.74616	60486.85	15353.96	264049.4	539282.8	8.915702
80 to 84	14217	1812	0.1275	5	1.2	0.106211	0.419631	0.580369	45132.9	18939.16	178316.6	275233.4	6.098288
85 and up	8605	2108	0.2450	3.7	1.2	0.204145	0.548271	0.451729	26193.74	14361.28	96916.82	96916.82	3.7
all ages	4836483	19909	0.0041										

2.2. Reduction to background level													
NCR (Male)	Clean Air Computations			Excess of 66.35 ug/m3PM10									
Age Grp	Population	Cases	Rate	N	RR	Clean Air A	nQx	nPx	nlx	ndx	Lx	Tx	Ex
Under 1	140,415	3634	0.0259	1	1.3	0.019908	0.019751	0.980249	100000	1975.074	98814.96	6767131	67.67131
1 to 4	487,282	1110	0.0023	4	1	0.002278	0.009079	0.990921	98024.93	889.9367	389963.9	6668316	68.02674
5 to 9	509576	421	0.0008	5	1	0.000826	0.004122	0.995878	97134.99	400.4264	484673.9	6278352	64.63533
10 to 14	438531	311	0.0007	5	1	0.000709	0.00354	0.99646	96734.56	342.4069	482816.8	5793678	59.89254
15 to 19	463906	570	0.0012	5	1	0.001229	0.006125	0.993875	96392.16	590.3704	480484.9	5310861	55.09641
20 to 24	496600	1180	0.0024	5	1	0.002376	0.011811	0.988189	95801.79	1131.479	476180.2	4830377	50.42053
25 to 29	487562	1414	0.0029	5	1	0.0029	0.014396	0.985604	94670.31	1362.906	469944.3	4354196	45.99326
30 to 34	397990	1380	0.0035	5	1.36	0.00255	0.012667	0.987333	93307.4	1181.938	463582.2	3884252	41.62855
35 to 39	337886	1458	0.0043	5	1.36	0.003173	0.015739	0.984261	92125.46	1449.996	457002.3	3420670	37.13056
40 to 44	260181	1539	0.0059	5	1.36	0.004349	0.021513	0.978487	90675.47	1950.685	448500.6	2963668	32.68434
45 to 49	200036	1854	0.0093	5	1.36	0.006815	0.033504	0.966496	88724.78	2972.629	436192.3	2515167	28.34796
50 to 54	129822	1763	0.0136	5	1.36	0.009985	0.048711	0.951289	85752.15	4177.07	418318.1	2078975	24.24399
55 to 59	98814	2074	0.0210	5	1.36	0.015433	0.074299	0.925701	81575.08	6060.91	392723.1	1660657	20.3574
60 to 64	70088	2204	0.0314	5	1.36	0.023122	0.109293	0.890707	75514.17	8253.187	356937.9	1267933	16.79067
65 to 69	45565	2172	0.0477	5	1.4	0.034049	0.156889	0.843111	67260.99	10552.5	309923.7	910995.5	13.54419
70 to 74	26416	1951	0.0739	5	1.4	0.052755	0.233039	0.766961	56708.49	13215.3	250504.2	601071.8	10.59933
75 to 79	14303	1594	0.1114	5	1.4	0.079604	0.331956	0.668044	43493.19	14437.83	181371.4	350567.7	8.060289
80 to 84	7664	1302	0.1699	5	1.4	0.121347	0.465512	0.534488	29055.36	13525.62	111462.7	169196.3	5.823239
85 and up	4,920	1165	0.2368	3.7	1.4	0.169135	0.476654	0.523346	15529.74	7402.31	57460.03	57733.55	3.717613
all ages	4,617,557	29096	0.0063										

2.2.Females				Excess PM10 of 66.35 ug/m3									
Age Grp	Population	Cases	Rate		RR	Clean Air A	nQx	nPx	nlx	ndx	Lx	Tx	Ex
Under 1	129201	2548	0.0197	1	1.3	0.01517	0.015079	0.984921	100000	1507.866	99095.28	7478781	74.78781
1 to 4	455632	877	0.0019	4	1	0.001925	0.007676	0.992324	98492.13	755.982	392154.2	7379686	74.92665
5 to 9	478433	308	0.0006	5	1	0.000644	0.003214	0.996786	97736.15	314.0917	487895.5	6987532	71.49383
10 to 14	433907	261	0.0006	5	1	0.000602	0.003003	0.996997	97422.06	292.5624	486378.9	6499636	66.71627
15 to 19	556091	304	0.0005	5	1	0.000547	0.00273	0.99727	97129.5	265.1281	484984.7	6013257	61.90969
20 to 24	581667	430	0.0007	5	1	0.000739	0.003689	0.996311	96864.37	357.3767	483428.4	5528273	57.0723
25 to 29	523779	510	0.0010	5	1	0.000974	0.004857	0.995143	96506.99	468.7	481363.2	5044844	52.27439
30 to 34	412827	620	0.0015	5	1.36	0.001104	0.005506	0.994494	96038.29	528.8126	478869.4	4563481	47.51731
35 to 39	340462	644	0.0019	5	1.36	0.001391	0.00693	0.99307	95509.48	661.8923	475892.7	4084612	42.76656
40 to 44	260845	758	0.0029	5	1.36	0.002137	0.010627	0.989373	94847.59	1007.93	471718.1	3608719	38.04756
45 to 49	198000	860	0.0043	5	1.36	0.003194	0.015842	0.984158	93839.66	1486.61	465481.8	3137001	33.42937
50 to 54	134158	898	0.0067	5	1.36	0.004922	0.02431	0.97569	92353.05	2245.076	456152.6	2671519	28.92724
55 to 59	109144	1074	0.0098	5	1.36	0.007235	0.035534	0.964466	90107.97	3201.94	442535	2215366	24.58569
60 to 64	82439	1297	0.0157	5	1.36	0.011568	0.056216	0.943784	86906.03	4885.47	422316.5	1772831	20.39941
65 to 69	57067	1440	0.0252	5	1.4	0.018024	0.086234	0.913766	82020.56	7072.957	392420.4	1350515	16.46557
70 to 74	37251	1545	0.0415	5	1.4	0.029625	0.137912	0.862088	74947.61	10336.19	348897.6	958094.6	12.78352
75 to 79	22758	1588	0.0698	5	1.4	0.049841	0.221595	0.778405	64611.42	14317.54	287263.2	609197	9.428628
80 to 84	14217	1812	0.1275	5	1.4	0.091038	0.370798	0.629202	50293.88	18648.86	204847.2	321933.8	6.401053
85 and up	8605	2108	0.2450	3.7	1.4	0.174981	0.489101	0.510899	31645.02	15477.62	117086.6	117086.6	3.7
all ages	4836483	19909	0.0041										

2.3. Reduction to 50 ug/m3													
NCR (Male)	Clean Air Computations	Pooled estimate											
Age Grp	Population	Cases	Rate	N	RR	Clean Air A	nQx	nPx	nlx	ndx	Lx	Tx	Ex
Under 1													
1 to 4	140,415	3634	0.0259	1	1.2	0.021567	0.021383	0.978617	100000	2138.256	98717.05	6672405	66.72405
5 to 9	487,282	1110	0.0023	4	1	0.002278	0.009079	0.990921	97861.74	888.4552	389314.7	6573688	67.17322
10 to 14	509576	421	0.0008	5	1	0.000826	0.004122	0.995878	96973.29	399.7599	483867	6184373	63.77399
15 to 19	438531	311	0.0007	5	1	0.000709	0.00354	0.99646	96573.53	341.8369	482013.1	5700506	59.02763
20 to 24	463906	570	0.0012	5	1	0.001229	0.006125	0.993875	96231.69	589.3876	479685	5218493	54.22843
25 to 29	496600	1180	0.0024	5	1	0.002376	0.011811	0.988189	95642.3	1129.596	475387.5	4738808	49.5472
30 to 34	487562	1414	0.0029	5	1.24	0.002339	0.011626	0.988374	94512.71	1098.819	469816.5	4263421	45.1095
35 to 39	397990	1380	0.0035	5	1.24	0.002796	0.013884	0.986116	93413.89	1297.004	463826.9	3793604	40.61071
40 to 44	337886	1458	0.0043	5	1.24	0.00348	0.017249	0.982751	92116.89	1588.96	456612	3329777	36.14731
45 to 49	260181	1539	0.0059	5	1.24	0.00477	0.02357	0.97643	90527.93	2133.759	447305.2	2873165	31.73789
50 to 54	200036	1854	0.0093	5	1.24	0.007474	0.036687	0.963313	88394.17	3242.897	433863.6	2425860	27.44367
55 to 59	129822	1763	0.0136	5	1.24	0.010952	0.053299	0.946701	85151.27	4538.503	414410.1	1991997	23.39362
60 to 64	98814	2074	0.0210	5	1.24	0.016927	0.081197	0.918803	80612.77	6545.5	386700.1	1577586	19.56993
65 to 69	70088	2204	0.0314	5	1.26	0.024957	0.117458	0.882542	74067.27	8699.783	348586.9	1190886	16.07844
70 to 74	45565	2172	0.0477	5	1.26	0.037832	0.172815	0.827185	65367.48	11296.46	298596.3	842299.5	12.8856
75 to 79	26416	1951	0.0739	5	1.26	0.058616	0.255623	0.744377	54071.03	13821.8	235600.6	543703.2	10.05535
80 to 84	14303	1594	0.1114	5	1.26	0.088449	0.362161	0.637839	40249.23	14576.7	164804.4	307902.6	7.649901
85 and up	7664	1302	0.1699	5	1.26	0.13483	0.504196	0.495804	25672.52	12943.99	96002.64	143098.2	5.573983
all ages	4,920	1165	0.2368	3.7	1.26	0.187927	0.515953	0.484047	12728.53	6567.319	47095.57	47095.57	3.7
	4,617,557	29096	0.0063										

2.3.Females				Excess PM10 of 45.6 ug/m3									
Age Grp	Population	Cases	Rate		RR	Clean Air A	nQx	nPx	nlx	ndx	Lx	Tx	Ex
Under 1	129201	2548	0.0197	1	1.2	0.016434	0.016327	0.983673	100000	1632.701	99020.38	7396151	73.96151
1 to 4	455632	877	0.0019	4	1	0.001925	0.007676	0.992324	98367.3	755.0239	391657.1	7297131	74.18249
5 to 9	478433	308	0.0006	5	1	0.000644	0.003214	0.996786	97612.27	313.6936	487277.1	6905474	70.74391
10 to 14	433907	261	0.0006	5	1	0.000602	0.003003	0.996997	97298.58	292.1916	485762.4	6418196	65.96393
15 to 19	556091	304	0.0005	5	1	0.000547	0.00273	0.99727	97006.39	264.792	484370	5932434	61.15509
20 to 24	581667	430	0.0007	5	1	0.000739	0.003689	0.996311	96741.6	356.9237	482815.7	5448064	56.31563
25 to 29	523779	510	0.0010	5	1.24	0.000785	0.003918	0.996082	96384.67	377.6823	480979.2	4965248	51.51492
30 to 34	412827	620	0.0015	5	1.24	0.001211	0.006038	0.993962	96006.99	579.6446	478585.8	4484269	46.70774
35 to 39	340462	644	0.0019	5	1.24	0.001525	0.007598	0.992402	95427.35	725.079	475324	4005683	41.97626
40 to 44	260845	758	0.0029	5	1.24	0.002344	0.011649	0.988351	94702.27	1103.211	470753.3	3530359	37.27851
45 to 49	198000	860	0.0043	5	1.24	0.003503	0.017362	0.982638	93599.06	1625.049	463932.7	3059606	32.68843
50 to 54	134158	898	0.0067	5	1.24	0.005398	0.026631	0.973369	91974.01	2449.354	453746.7	2595673	28.22181
55 to 59	109144	1074	0.0098	5	1.24	0.007936	0.038906	0.961094	89524.65	3483.082	438915.6	2141927	23.92555
60 to 64	82439	1297	0.0157	5	1.26	0.012486	0.060542	0.939458	86041.57	5209.133	417185	1703011	19.79289
65 to 69	57067	1440	0.0252	5	1.26	0.020027	0.095359	0.904641	80832.44	7708.073	384892	1285826	15.9073
70 to 74	37251	1545	0.0415	5	1.26	0.032917	0.152071	0.847929	73124.37	11120.07	337821.7	900934.1	12.32057
75 to 79	22758	1588	0.0698	5	1.26	0.055379	0.243222	0.756778	62004.3	15080.81	272319.5	563112.5	9.08183
80 to 84	14217	1812	0.1275	5	1.26	0.101153	0.403682	0.596318	46923.49	18942.16	187262.1	290793	6.197173
85 and up	8805	2108	0.2450	3.7	1.26	0.194424	0.52907	0.47093	27981.33	14804.08	103530.9	103530.9	3.7
all ages	4836483	19909	0.0041										

Life Expectancy Tables 3 :Comparison of Pollution Scenarios using two relative risk coefficients,
1.04 - infants, 1.05- 30 y/o and above, for Metropolitan Manila, 1995
3.1. Reduction to 60 ug/m3

NCR (Male)	Clean Air Computations	Pooled estimate				Clean Air ASDR	nQx	nPx	nlx	ndx	Lx	Tx	Ex
Age Grp	Population	Cases	Rate	N	RR								
Under 1	140,415	3634	0.0259	1	1.15	0.0225	0.0223	0.9777	100000	2230.39	98661.76	6605372	66.05372
1 to 4	487,282	1110	0.0023	4	1	0.0023	0.0091	0.9909	97769.61	887.62	388948.1	6506710	66.55146
5 to 9	509576	421	0.0008	5	1	0.0008	0.0041	0.9959	96881.99	399.38	483411.5	6117762	63.14654
10 to 14	438531	311	0.0007	5	1	0.0007	0.0035	0.9965	96482.6	341.52	481559.2	5634351	58.39758
15 to 19	463906	570	0.0012	5	1	0.0012	0.0061	0.9939	96141.09	588.83	479233.4	5152791	53.59614
20 to 24	496600	1180	0.0024	5	1	0.0024	0.0118	0.9882	95552.26	1128.53	474939.9	4673558	48.91102
25 to 29	487562	1414	0.0029	5	1	0.0029	0.0144	0.9856	94423.72	1359.36	468720.2	4198618	44.46571
30 to 34	397990	1380	0.0035	5	1.19	0.0029	0.0145	0.9855	93064.37	1346.05	461956.7	3729898	40.07869
35 to 39	337886	1458	0.0043	5	1.19	0.0036	0.0180	0.9820	91718.32	1647.96	454471.7	3267941	35.63019
40 to 44	260181	1539	0.0059	5	1.19	0.0050	0.0245	0.9755	90070.36	2211.08	444824.1	2813469	31.23635
45 to 49	200036	1854	0.0093	5	1.19	0.0078	0.0382	0.9618	87859.28	3356.12	430906.1	2368645	26.95954
50 to 54	129822	1763	0.0136	5	1.19	0.0114	0.0555	0.9445	84503.16	4687.95	410795.9	1937739	22.93097
55 to 59	98814	2074	0.0210	5	1.19	0.0176	0.0845	0.9155	79815.21	6741.54	382222.2	1526943	19.13098
60 to 64	70088	2204	0.0314	5	1.19	0.0264	0.1239	0.8761	73073.66	9056.68	342726.6	1144721	15.6653
65 to 69	45565	2172	0.0477	5	1.19	0.0401	0.1821	0.8179	64016.99	11654.60	290948.4	801994.5	12.52784
70 to 74	26416	1951	0.0739	5	1.19	0.0621	0.2686	0.7314	52362.39	14066.63	226645.4	511046	9.759793
75 to 79	14303	1594	0.1114	5	1.19	0.0937	0.3794	0.6206	38295.76	14530.30	155153	284400.7	7.426428
80 to 84	7664	1302	0.1699	5	1.19	0.1428	0.5261	0.4739	23765.46	12501.91	87572.52	129247.6	5.438466
85 and up	4,920	1165	0.2368	3.7	1.19	0.1990	0.5381	0.4619	11263.55	6061.33	41675.13	41675.13	3.7
all ages	4,617,557	29096	0.0063										

3.1.Females				Excess PM10 of 35.6ug/m3									
Age Grp	Population	Cases	Rate		RR	Clean Air ASDR	nQx	nPx	nlx	ndx	Lx	Tx	Ex
Under 1	129201	2548	0.0197	1	1.15	0.017149	0.017032	0.982968	100000	1703.205	98978.08	7344218	73.44218
1 to 4	455632	877	0.0019	4	1	0.001925	0.007676	0.992324	98296.8	754.4827	391376.4	7245240	73.7078
5 to 9	478433	308	0.0006	5	1	0.000644	0.003214	0.996786	97542.31	313.4687	486927.9	6853864	70.26555
10 to 14	433907	261	0.0006	5	1	0.000602	0.003003	0.996997	97228.84	291.9822	485414.3	6366936	65.48402
15 to 19	556091	304	0.0005	5	1	0.000547	0.00273	0.99727	96936.86	264.6023	484022.8	5881522	60.67374
20 to 24	581667	430	0.0007	5	1	0.000739	0.003689	0.996311	96672.26	356.6679	482469.6	5397499	55.83297
25 to 29	523779	510	0.0010	5	1	0.000974	0.004857	0.995143	96315.59	467.7705	480408.5	4915029	51.03046
30 to 34	412827	620	0.0015	5	1.19	0.001262	0.00629	0.99371	95847.82	602.9215	477731.8	4434621	46.26731
35 to 39	340462	644	0.0019	5	1.19	0.00159	0.007916	0.992084	95244.9	753.9796	474339.5	3956889	41.54437
40 to 44	260845	758	0.0029	5	1.19	0.002442	0.012136	0.987864	94490.92	1146.718	469587.8	3482549	36.85592
45 to 49	198000	860	0.0043	5	1.19	0.00365	0.018085	0.981915	93344.2	1688.102	462500.8	3012962	32.27797
50 to 54	134158	898	0.0067	5	1.19	0.005625	0.027734	0.972266	91656.1	2542.024	451925.4	2550461	27.82642
55 to 59	109144	1074	0.0098	5	1.19	0.008269	0.040508	0.959492	89114.08	3609.834	436545.8	2098535	23.54887
60 to 64	82439	1297	0.0157	5	1.19	0.013221	0.063989	0.936011	85504.24	5471.365	413842.8	1661990	19.43751
65 to 69	57067	1440	0.0252	5	1.19	0.021205	0.100686	0.899314	80032.88	8058.158	380019	1248147	15.59543
70 to 74	37251	1545	0.0415	5	1.19	0.034853	0.160299	0.839701	71974.72	11537.48	331029.9	868127.8	12.06157
75 to 79	22758	1588	0.0698	5	1.19	0.058637	0.2557	0.7443	60437.24	15453.8	263551.7	537097.9	8.88687
80 to 84	14217	1812	0.1275	5	1.19	0.107103	0.422412	0.577588	44983.44	19001.57	177413.3	273546.2	6.081043
85 and up	8805	2108	0.2450	3.7	1.19	0.20586	0.551608	0.448392	25981.88	14331.81	96132.94	96132.94	3.7
all ages	4836483	19909	0.0041										

3.2. Reduction to background level													
NCR (Male)	Clean Air. Computations			Excess of 66.35 ug/m3PM10									
Age Grp	Population	Cases	Rate	N	RR	Clean Air A	nQx	nPx	nlx	ndx	Lx	Tx	Ex
Under 1	140,415	3634	0.0259	1	1.3	0.019908	0.019751	0.980249	100000	1975.074	98814.96	6769064	67.69064
1 to 4	487,282	1110	0.0023	4	1	0.002278	0.009079	0.990921	98024.93	889.9367	389963.9	6670249	68.04648
5 to 9	509576	421	0.0008	5	1	0.000826	0.004122	0.995878	97134.99	400.4264	484673.9	6280285	64.65523
10 to 14	438531	311	0.0007	5	1	0.000709	0.00354	0.99646	96734.56	342.4069	482816.8	5795612	59.91252
15 to 19	463906	570	0.0012	5	1	0.001229	0.006125	0.993875	96392.16	590.3704	480484.9	5312795	55.11646
20 to 24	496600	1180	0.0024	5	1	0.002376	0.011811	0.988189	95801.79	1131.479	476180.2	4832310	50.44071
25 to 29	487562	1414	0.0029	5	1	0.0029	0.014396	0.985604	94670.31	1362.906	469944.3	4356130	46.01369
30 to 34	397990	1380	0.0035	5	1.38	0.002513	0.012485	0.987515	93307.4	1164.915	463624.7	3886185	41.64927
35 to 39	337886	1458	0.0043	5	1.38	0.003127	0.015513	0.984487	92142.48	1429.409	457138.9	3422561	37.14422
40 to 44	260181	1539	0.0059	5	1.38	0.004286	0.021204	0.978796	90713.08	1923.512	448756.6	2965422	32.69013
45 to 49	200036	1854	0.0093	5	1.38	0.006716	0.033026	0.966974	88789.56	2932.398	436616.8	2516665	28.34416
50 to 54	129822	1763	0.0136	5	1.38	0.009841	0.048022	0.951978	85857.17	4123.029	418978.3	2080048	24.22685
55 to 59	98814	2074	0.0210	5	1.38	0.015209	0.073261	0.926739	81734.14	5987.941	393700.8	1661070	20.32284
60 to 64	70088	2204	0.0314	5	1.38	0.022787	0.107795	0.892205	75746.2	8165.033	358318.4	1267369	16.73179
65 to 69	45565	2172	0.0477	5	1.38	0.034542	0.158982	0.841018	67581.16	10744.18	311045.4	909050.9	13.45125
70 to 74	26416	1951	0.0739	5	1.38	0.053519	0.236018	0.763982	56836.99	13414.56	250648.5	598005.5	10.52141
75 to 79	14303	1594	0.1114	5	1.38	0.080757	0.335959	0.664041	43422.43	14588.15	180641.8	347357	7.999482
80 to 84	7664	1302	0.1699	5	1.38	0.123105	0.470671	0.529329	28834.28	13571.46	110242.8	166715.2	5.78184
85 and up	4,920	1165	0.2368	3.7	1.38	0.171586	0.481897	0.518103	15262.82	7355.115	56472.44	56472.44	3.7
all ages	4,617,557	29096	0.0063										

3.2.Females				Excess PM10 of 66.35 ug/m3									
Age Grp	Population	Cases	Rate		RR	Clean Air A	nQx	nPx	nlx	ndx	Lx	Tx	Ex
Under 1	129201	2548	0.0197	1	1.3	0.01517	0.015079	0.984921	100000	1507.866	99095.28	7477775	74.77775
1 to 4	455632	877	0.0019	4	1	0.001925	0.007676	0.992324	98492.13	755.982	392154.2	7378680	74.91644
5 to 9	478433	308	0.0006	5	1	0.000644	0.003214	0.996786	97736.15	314.0917	487895.5	6986526	71.48353
10 to 14	433907	261	0.0006	5	1	0.000602	0.003003	0.996997	97422.06	292.5624	486378.9	6498630	66.70594
15 to 19	556091	304	0.0005	5	1	0.000547	0.00273	0.99727	97129.5	265.1281	484984.7	6012251	61.89933
20 to 24	581667	430	0.0007	5	1	0.000739	0.003689	0.996311	96864.37	357.3767	483428.4	5527266	57.06192
25 to 29	523779	510	0.0010	5	1	0.000974	0.004857	0.995143	96506.99	468.7	481363.2	5043838	52.26396
30 to 34	412827	620	0.0015	5	1.38	0.001088	0.005427	0.994573	96038.29	521.1695	478888.5	4562475	47.50683
35 to 39	340462	644	0.0019	5	1.38	0.001371	0.00683	0.99317	95517.12	652.3846	475954.7	4083586	42.7524
40 to 44	260845	758	0.0029	5	1.38	0.002106	0.010474	0.989526	94864.74	993.5784	471839.7	3607632	38.02922
45 to 49	198000	860	0.0043	5	1.38	0.003147	0.015614	0.984386	93871.16	1465.725	465691.5	3135792	33.40527
50 to 54	134158	898	0.0067	5	1.38	0.00485	0.023962	0.976038	92405.44	2214.183	456491.7	2670100	28.89549
55 to 59	109144	1074	0.0098	5	1.38	0.007131	0.035029	0.964971	90191.25	3159.265	443058.1	2213609	24.5435
60 to 64	82439	1297	0.0157	5	1.38	0.011401	0.055423	0.944577	87031.99	4823.609	423100.9	1770551	20.34368
65 to 69	57067	1440	0.0252	5	1.38	0.018285	0.087429	0.912571	82208.38	7187.403	393073.4	1347450	16.39066
70 to 74	37251	1545	0.0415	5	1.38	0.030055	0.139771	0.860229	75020.97	10485.77	348890.4	954376.3	12.72146
75 to 79	22758	1588	0.0698	5	1.38	0.050564	0.224446	0.775554	64535.2	14484.65	286464.4	605485.9	9.382257
80 to 84	14217	1812	0.1275	5	1.38	0.092357	0.375164	0.624836	50050.55	18777.15	203309.9	319021.5	6.373985
85 and up	8605	2108	0.2450	3.7	1.38	0.177517	0.494437	0.505563	31273.4	15462.74	115711.6	115711.6	3.7
all ages	4836483	19909	0.0041										

3.3. Reduction to 50 ug/m3													
NCR (Male)	Clean Air Computations			Excess of 45.6 ug/m3PM10									
Age Grp	Population	Cases	Rate	N	RR	Clean Air A	nQx	nPx	nlx	ndx	Lx	Tx	Ex
Under 1													
1 to 4	140,415	3634	0.0259	1	1.2	0.021567	0.021383	0.978617	100000	2138.256	98717.05	6672225	66.72225
5 to 9	487,282	1110	0.0023	4	1	0.002278	0.009079	0.990921	97861.74	888.4552	389314.7	6573508	67.17138
10 to 14	509576	421	0.0008	5	1	0.000826	0.004122	0.995878	96973.29	399.7599	483867	6184194	63.77214
15 to 19	438531	311	0.0007	5	1	0.000709	0.00354	0.99646	96573.53	341.8369	482013.1	5700327	59.02577
20 to 24	463906	570	0.0012	5	1	0.001229	0.006125	0.993875	96231.69	589.3876	479685	5218314	54.22656
25 to 29	496600	1180	0.0024	5	1	0.002376	0.011811	0.988189	95642.3	1129.596	475387.5	4738629	49.54532
30 to 34	487562	1414	0.0029	5	1.25	0.00232	0.011534	0.988466	94512.71	1090.079	469838.3	4263241	45.1076
35 to 39	397990	1380	0.0035	5	1.25	0.002774	0.013774	0.986226	93422.63	1286.82	463896.1	3793403	40.60475
40 to 44	337886	1458	0.0043	5	1.25	0.003452	0.017113	0.982887	92135.81	1576.681	456737.3	3329507	36.13694
45 to 49	260181	1539	0.0059	5	1.25	0.004732	0.023384	0.976616	90559.13	2117.618	447501.6	2872769	31.72258
50 to 54	200036	1854	0.0093	5	1.25	0.007415	0.036399	0.963601	88441.51	3219.149	434159.7	2425268	27.42228
55 to 59	129822	1763	0.0136	5	1.25	0.010864	0.052884	0.947116	85222.36	4506.915	414844.5	1991108	23.36368
60 to 64	98814	2074	0.0210	5	1.25	0.016791	0.080573	0.919427	80715.45	6503.519	387318.4	1576263	19.52865
65 to 69	70088	2204	0.0314	5	1.25	0.025157	0.118342	0.881658	74211.93	8782.382	349103.7	1188945	16.02094
70 to 74	45565	2172	0.0477	5	1.25	0.038135	0.174077	0.825923	65429.55	11389.77	298673.3	839841.4	12.83581
75 to 79	26416	1951	0.0739	5	1.25	0.059085	0.257405	0.742595	54039.78	13910.1	235423.6	541168	10.01425
80 to 84	14303	1594	0.1114	5	1.25	0.089156	0.36453	0.63547	40129.68	14628.48	164077.2	305744.4	7.61891
85 and up	7664	1302	0.1699	5	1.25	0.135908	0.507207	0.492793	25501.19	12934.38	95170.02	141667.2	5.555317
all ages	4,920	1165	0.2368	3.7	1.25	0.189431	0.519009	0.480991	12566.81	6522.29	46497.21	46497.21	3.7
	4,617,557	29096	0.0063										

3.3.Females				Excess PM10 of 45.6 ug/m3									
Age Grp	Population	Cases	Rate		RR	Clean Air A	nQx	nPx	nlx	ndx	Lx	Tx	Ex
Under 1	129201	2548	0.0197	1	1.2	0.016434	0.016327	0.983673	100000	1632.701	99020.38	7395186	73.95186
1 to 4	455632	877	0.0019	4	1	0.001925	0.007676	0.992324	98367.3	755.0239	391657.1	7296166	74.17268
5 to 9	478433	308	0.0006	5	1	0.000644	0.003214	0.996786	97612.27	313.6936	487277.1	6904509	70.73402
10 to 14	433907	261	0.0006	5	1	0.000602	0.003003	0.996997	97298.58	292.1916	485762.4	6417232	65.95401
15 to 19	556091	304	0.0005	5	1	0.000547	0.00273	0.99727	97006.39	264.792	484370	5931469	61.14514
20 to 24	581667	430	0.0007	5	1	0.000739	0.003689	0.996311	96741.6	356.9237	482815.7	5447099	56.30566
25 to 29	523779	510	0.0010	5	1.25	0.000779	0.003887	0.996113	96384.67	374.6667	480986.7	4964283	51.5049
30 to 34	412827	620	0.0015	5	1.25	0.001201	0.005989	0.994011	96010.01	575.0393	478612.4	4483297	46.69614
35 to 39	340462	644	0.0019	5	1.25	0.001513	0.007538	0.992462	95434.97	719.3577	475376.4	4004684	41.96244
40 to 44	260845	758	0.0029	5	1.25	0.002325	0.011557	0.988443	94715.61	1094.59	470841.6	3529308	37.26216
45 to 49	198000	860	0.0043	5	1.25	0.003475	0.017224	0.982776	93621.02	1612.539	464073.8	3058466	32.66859
50 to 54	134158	898	0.0067	5	1.25	0.005355	0.026421	0.973579	92008.48	2430.929	453965.1	2594393	28.19732
55 to 59	109144	1074	0.0098	5	1.25	0.007872	0.038601	0.961399	89577.55	3457.797	439243.3	2140427	23.89469
60 to 64	82439	1297	0.0157	5	1.25	0.012586	0.061012	0.938988	86119.76	5254.305	417463	1701184	19.7537
65 to 69	57067	1440	0.0252	5	1.25	0.020187	0.096085	0.903915	80865.45	7769.947	384902.4	1283721	15.87478
70 to 74	37251	1545	0.0415	5	1.25	0.03318	0.153194	0.846806	73095.5	11197.79	337483	898818.8	12.2965
75 to 79	22758	1588	0.0698	5	1.25	0.055822	0.244929	0.755071	61897.71	15160.57	271587.1	561335.7	9.068764
80 to 84	14217	1812	0.1275	5	1.25	0.101962	0.406255	0.593745	46737.14	18987.21	186217.7	289748.6	6.199537
85 and up	8605	2108	0.2450	3.7	1.25	0.195979	0.532176	0.467824	27749.93	14767.85	102674.7	103530.9	3.730854
all ages	4836483	19909	0.0041										

Life Expectancy Table 4.1:Estimates for different socio-economic classes according to Educational attainment in Metropolitan Manila, Philippines, using the US adult mortality rate ratio estimates, 1995

SES: high.middle and lower educational levels

Males	Population	Deaths	DR		Mx-LSES	Mx-MSES	Mx-HSES	Qx-LSES	Qx-MSES	Qx-HSES	Px-LSES	Px-MSES
All Ages					28%	49%	23%					
25 to 29	487562	1414	0.0029	5	0.004175	0.002799	0.001564	0.020659	0.013898	0.007788	0.979341	0.986102
30 to 34	397990	1380	0.0035	5	0.004992	0.003346	0.00187	0.024651	0.016594	0.009304	0.975349	0.983406
35 to 39	337886	1458	0.0043	5	0.006212	0.004165	0.002327	0.030585	0.020608	0.011566	0.969415	0.979392
40 to 44	260181	1539	0.0059	5	0.008515	0.005709	0.003189	0.041689	0.028142	0.01582	0.958311	0.971858
45 to 49	200036	1854	0.0093	5	0.01188	0.011198	0.005894	0.057687	0.054465	0.02904	0.942313	0.945535
50 to 54	129822	1763	0.0136	5	0.017407	0.016407	0.008635	0.083405	0.078804	0.042265	0.916595	0.921196
55 to 59	98814	2074	0.0210	5	0.026903	0.025359	0.013347	0.12604	0.119234	0.064578	0.87396	0.880766
60 to 64	70088	2204	0.0314	5	0.040307	0.037993	0.019996	0.183088	0.173487	0.095221	0.816912	0.826513
65 to 69	45565	2172	0.0477	5	0.053754	0.053754	0.040115	0.236928	0.236928	0.182292	0.763072	0.763072
70 to 74	26416	1951	0.0739	5	0.083285	0.083285	0.062153	0.344663	0.344663	0.268973	0.655337	0.655337
75 to 79	14303	1594	0.1114	5	0.125672	0.125672	0.093785	0.47814	0.47814	0.379863	0.52186	0.52186
80 to 84	7664	1302	0.1699	5	0.191573	0.191573	0.142965	0.647673	0.647673	0.526608	0.352327	0.352327
85 and up	4,920	1165	0.2368	3.7	0.267017	0.267017	0.199267	0.661296	0.661296	0.538699	0.338704	0.338704
	4,617,557											

Px-HSES	lx-LSES	lx-MSES	lx-HSES	dx-LSES	dx-MSES	dx-HSES	Lx-LSES	Lx-MSES	Lx-HSES	Tx-LSES	Tx-MSES	Tx-HSES
0.992212	100,000	100,000	100,000	2065.94	1389.762	778.792	494835.2	496525.6	498053	3981991	4128738	4656594
0.990696	97934.06	98610.24	99221.21	2414.139	1636.289	923.1721	483635	488960.5	493798.1	3487156	3632212	4158541
0.988434	95519.92	96973.95	98298.04	2921.435	1998.451	1136.867	470296	479873.6	488648	3003521	3143252	3664743
0.98418	92598.49	94975.5	97161.17	3860.345	2672.819	1537.104	453341.6	468195.4	481963.1	2533225	2663378	3176095
0.97096	88738.14	92302.68	95624.06	5119.041	5027.248	2776.951	430893.1	448945.3	471177.9	2079884	2195183	2694131
0.957735	83619.1	87275.43	92847.11	6974.248	6877.695	3924.173	400659.9	419182.9	454425.1	1648990	1746237	2222953
0.935422	76644.85	80397.74	88922.94	9660.311	9586.149	5742.505	359073.5	378023.3	430258.4	1248331	1327054	1768528
0.904779	66984.54	70811.59	83180.44	12264.05	12284.87	7920.551	304262.6	323345.8	396100.8	889257.1	949031.1	1338270
0.817708	54720.49	58526.72	75259.89	12964.84	13866.64	13719.24	241190.4	257967	342001.3	584994.5	625685.4	942169.1
0.731027	41755.65	44660.08	61540.64	14391.65	15392.7	16552.75	172799.1	184818.7	266321.3	343804.1	367718.4	600167.8
0.620137	27364.01	29267.38	44987.9	13083.81	13993.89	17089.23	104110.5	111352.2	182216.4	171005	182899.7	333846.4
0.473392	14280.19	15273.49	27898.66	9248.896	9892.228	14691.67	48278.72	51636.87	102764.2	66894.51	71547.53	151630
0.461301	5031.295	5381.26	13207	3327.175	3558.605	7114.596	18615.79	19910.66	48865.89	18615.79	19910.66	48865.89

Ex-LSES	Ex-MSES	Ex-HSES
39.81991	41.28738	46.56594
35.60718	36.83403	41.91181
31.44392	32.41336	37.28195
27.35709	28.04279	32.68893
23.43844	23.78244	28.1742
19.72026	20.00835	23.94209
16.28721	16.50612	19.88832
13.27556	13.4022	16.08876
10.69059	10.69059	12.51888
8.233715	8.233715	9.752381
6.249268	6.249268	7.420806
4.684427	4.684427	5.435029
3.7	3.7	3.7

Life Expectancy Table 4.2:Estimates for different socio-economic classes according to Educational attainment in Metropolitan Manila, *Philippines*, using the US adult mortality rate ratio estimates, 1995

FEMALES	SES: high, middle and low educational groups											
NCR												
All Ages	Population	Cases	Rate	N	Mx-LSES	Mx-MSES	Mx-HSES	Qx-LSES	Qx-MSES	Qx-HSES	Px-LSES	Px-MSES
					33%	43%	24%					
25 to 29	523779	510	0.0010	5	0.001336	0.000882	5.64E-05	0.006658	0.004401	0.000282	0.993342	0.995599
30 to 34	412827	620	0.0015	5	0.002061	0.001361	8.7E-05	0.010251	0.006781	0.000435	0.989749	0.993219
35 to 39	340462	644	0.0019	5	0.002596	0.001714	0.00011	0.012894	0.008533	0.000547	0.987106	0.991467
40 to 44	260845	758	0.0029	5	0.003988	0.002633	0.000168	0.019741	0.013079	0.000841	0.980259	0.986921
45 to 49	198000	860	0.0043	5	0.00553	0.003998	0.003331	0.027273	0.01979	0.016519	0.972727	0.98021
50 to 54	134158	898	0.0067	5	0.008522	0.006161	0.005134	0.041723	0.030336	0.025344	0.958277	0.969664
55 to 59	109144	1074	0.0098	5	0.012529	0.009057	0.007547	0.06074	0.044281	0.037038	0.93926	0.955719
60 to 64	82439	1297	0.0157	5	0.020031	0.01448	0.012067	0.095379	0.069872	0.058568	0.904621	0.930128
65 to 69	57067	1440	0.0252	5	0.027556	0.035353	0.022774	0.128902	0.16241	0.107736	0.871098	0.83759
70 to 74	37251	1545	0.0415	5	0.045294	0.058108	0.037433	0.203432	0.253688	0.171147	0.796568	0.746312
75 to 79	22758	1588	0.0698	5	0.076201	0.09776	0.062976	0.320038	0.392801	0.27205	0.679962	0.607199
80 to 84	14217	1812	0.1275	5	0.139186	0.178565	0.11503	0.516282	0.617269	0.446692	0.483718	0.382731
85 and up	8605	2108	0.2450	3.7	0.267526	0.343215	0.221096	0.662138	0.77672	0.580581	0.337862	0.22328

Px-HSES	lx-LSES	lx-MSES	lx-HSES	dx-LSES	dx-MSES	dx-HSES	Lx-LSES	Lx-MSES	Lx-HSES	Tx-LSES	Tx-MSES	Tx-HSES
0.999718	100,000	100,000	100,000	665.8275	440.1351	28.18682	498335.4	498899.7	499929.5	4772356	4825194	5212032
0.999565	99334.17	99559.86	99971.81	1018.306	675.0776	43.46022	494125.1	496111.6	499750.4	4274021	4326294	4712103
0.999453	98315.87	98884.79	99928.35	1267.709	843.7438	54.7107	488410.1	492314.6	499505	3779895	3830182	4212352
0.999159	97048.16	98041.04	99873.64	1915.816	1282.23	83.99243	480451.2	486999.6	499158.2	3291485	3337868	3712847
0.983481	95132.34	96758.81	99789.65	2594.571	1914.896	1648.45	469175.3	479006.8	494827.1	2811034	2850868	3213689
0.974656	92537.77	94843.92	98141.2	3860.914	2877.21	2487.319	453036.6	467026.6	484487.7	2341859	2371861	2718862
0.962962	88676.86	91966.71	95653.88	5386.266	4072.412	3542.811	429918.6	449652.5	469412.4	1888822	1904835	2234374
0.941432	83290.59	87894.29	92111.07	7944.176	6141.357	5394.738	396592.5	424118.1	447068.5	1458904	1455182	1764962
0.892264	75346.41	81752.94	86716.33	9712.302	13277.5	9342.442	352451.3	375570.9	410225.6	1062311	1031064	1317893
0.828853	65634.11	68475.44	77373.89	13352.09	17371.37	13242.32	294790.3	298948.8	353763.7	709859.9	655493.3	907667.7
0.72795	52282.02	51104.06	64131.57	16732.23	20073.74	17446.96	219579.5	205336	277040.5	415069.6	356544.5	553904
0.553308	35549.79	31030.32	46684.61	18353.72	19154.07	20853.64	131864.6	107266.4	181289	195490.1	151208.6	276863.6
0.419419	17196.06	11876.25	25830.97	11386.17	9224.521	14996.96	63625.44	43942.14	95574.6	63625.44	43942.14	95574.6

Ex-LSES	Ex-MSES	Ex-HSES
47.72356	48.25194	52.12032
43.02669	43.4542	47.13431
38.44644	38.73379	42.15372
33.916	34.04562	37.17545
29.54867	29.46365	32.20463
25.30706	25.00805	27.70357
21.30006	20.71222	23.35895
17.51583	16.55605	19.16123
14.09903	12.61195	15.19775
10.81541	9.572678	11.73093
7.939051	6.976834	8.636994
5.49905	4.87293	5.93051
3.7	3.7	3.7

Life Expectancy Tables 5: Comparison of Pollution Reduction Scenarios using relative risk coefficients of 1.35, 1.23, and 1.06 respective

5.1. Reduction to 60 ug/m3

FEMALES	**SES: high, middle and low educational groups**											
All Ages	Population	Cases	Rate	N		Mx-LSES	clean-l		Mx-MSES			Mx-HSES
25 to 29	523779	510	0.0010	5	1	0.001336	0.001336	1	0.000882	0.000882	1	5.64E-05
30 to 34	412827	620	0.0015	5	1.36	0.002061	0.001515	1.23	0.001361	0.001106	1.06	8.7E-05
35 to 39	340462	644	0.0019	5	1.36	0.002596	0.001909	1.23	0.001714	0.001393	1.06	0.00011
40 to 44	260845	758	0.0029	5	1.36	0.003988	0.002932	1.23	0.002633	0.002141	1.06	0.000168
45 to 49	198000	860	0.0043	5	1.36	0.00553	0.004066	1.23	0.003998	0.00325	1.06	0.003331
50 to 54	134158	898	0.0067	5	1.36	0.008522	0.006266	1.23	0.006161	0.005009	1.06	0.005134
55 to 59	109144	1074	0.0098	5	1.36	0.012529	0.009212	1.23	0.009057	0.007363	1.06	0.007547
60 to 64	82439	1297	0.0157	5	1.36	0.020031	0.014729	1.23	0.01448	0.011773	1.06	0.012067
65 to 69	57067	1440	0.0252	5	1.36	0.027556	0.020262	1.23	0.035353	0.028742	1.06	0.022774
70 to 74	37251	1545	0.0415	5	1.36	0.045294	0.033304	1.23	0.058108	0.047242	1.06	0.037433
75 to 79	22758	1588	0.0698	5	1.36	0.076201	0.05603	1.23	0.09776	0.07948	1.06	0.062976
80 to 84	14217	1812	0.1275	5	1.36	0.139186	0.102343	1.23	0.178565	0.145175	1.06	0.11503
85 and up	8605	2108	0.2450	3.7	1.36	0.267526	0.19671	1.23	0.343215	0.279037	1.06	0.221096

Life Expectancy Tables 5: Comparison of Pollution Reduction Scenarios using relative risk coefficients of 1.35, 1.23, and 1.06 respective												
5.1. Reduction to 60 ug/m3												
FEMALES	SES: high, middle and low educational groups											
All Ages	Population	Cases	Rate	N		Mx-LSES	clean-I		Mx-MSES			Mx-HSES
25 to 29	523779	510	0.0010	5	1	0.001336	0.001336	1	0.000882	0.000882	1	5.64E-05
30 to 34	412827	620	0.0015	5	1.36	0.002061	0.001515	1.23	0.001361	0.001106	1.06	8.7E-05
35 to 39	340462	644	0.0019	5	1.36	0.002596	0.001909	1.23	0.001714	0.001393	1.06	0.00011
40 to 44	260845	758	0.0029	5	1.36	0.003988	0.002932	1.23	0.002633	0.002141	1.06	0.000168
45 to 49	198000	860	0.0043	5	1.36	0.00553	0.004066	1.23	0.003998	0.00325	1.06	0.003331
50 to 54	134158	898	0.0067	5	1.36	0.008522	0.006266	1.23	0.006161	0.005009	1.06	0.005134
55 to 59	109144	1074	0.0098	5	1.36	0.012529	0.009212	1.23	0.009057	0.007363	1.06	0.007547
60 to 64	82439	1297	0.0157	5	1.36	0.020031	0.014729	1.23	0.01448	0.011773	1.06	0.012067
65 to 69	57067	1440	0.0252	5	1.36	0.027556	0.020262	1.23	0.035353	0.028742	1.06	0.022774
70 to 74	37251	1545	0.0415	5	1.36	0.045294	0.033304	1.23	0.058108	0.047242	1.06	0.037433
75 to 79	22758	1588	0.0698	5	1.36	0.076201	0.05603	1.23	0.09776	0.07948	1.06	0.062976
80 to 84	14217	1812	0.1275	5	1.36	0.139186	0.102343	1.23	0.178565	0.145175	1.06	0.11503
85 and up	8605	2108	0.2450	3.7	1.36	0.267526	0.19671	1.23	0.343215	0.279037	1.06	0.221096

ly for low, middle and high educational levels

-	Qx-LSES	Qx-MSES	Qx-HSES	Px-LSES	Px-MSES	Px-HSES	lx-LSES	lx-MSES	lx-HSES	dx-LSES	dx-MSES	dx-HSES
5.64E-05	0.006658	0.004401	0.000282	0.993342	0.995599	0.999718	100,000	100,000	100,000	665.8275	440.1351	28.18682
8.2E-05	0.007548	0.005516	0.00041	0.992452	0.994484	0.99959	99334.17	99559.86	99971.81	749.7718	549.1917	41.00072
0.000103	0.009497	0.006943	0.000517	0.990503	0.993057	0.999483	98584.4	99010.67	99930.81	936.2831	687.3922	51.61594
0.000159	0.014553	0.010646	0.000793	0.985447	0.989354	0.999207	97648.12	98323.28	99879.2	1421.11	1046.745	79.24443
0.003143	0.020127	0.01612	0.015591	0.979873	0.98388	0.984409	96227.01	97276.54	99799.95	1936.716	1568.058	1556.03
0.004843	0.030849	0.024734	0.023927	0.969151	0.975266	0.976073	94290.29	95708.48	98243.92	2908.734	2367.233	2350.67
0.00712	0.045024	0.036151	0.034978	0.954976	0.963849	0.965022	91381.56	93341.25	95893.25	4114.363	3374.359	3354.154
0.011384	0.071028	0.05718	0.055344	0.928972	0.94282	0.944656	87267.19	89966.89	92539.1	6198.439	5144.316	5121.514
0.021485	0.096426	0.134077	0.101948	0.903574	0.865923	0.898052	81068.76	84822.57	87417.58	7817.132	11372.72	8912.072
0.035314	0.153721	0.211261	0.162245	0.846279	0.788739	0.837755	73251.62	73449.85	78505.51	11260.35	15517.09	12737.16
0.059412	0.245731	0.331526	0.258642	0.754269	0.668474	0.741358	61991.28	57932.76	65768.35	15233.16	19206.22	17010.45
0.108519	0.407462	0.532581	0.426803	0.592538	0.467419	0.573197	46758.12	38726.54	48757.9	19052.14	20625.04	20810.03
0.208581	0.533631	0.680929	0.556868	0.466369	0.319071	0.443132	27705.97	18101.5	27947.87	14784.78	12325.83	15563.27

Lx-LSES	Lx-MSES	Lx-HSES	Tx-LSES	Tx-MSES	Tx-HSES	Ex-LSES	Ex-MSES	Ex-HSES
498335.4	498899.7	499929.5	5060790	5002823	5256814	50.6079	50.02823	52.56814
494796.4	496426.3	499756.6	4562454	4503923	4756884	45.93036	45.23834	47.58225
490581.3	493334.9	499525	4067658	4007497	4257128	41.26066	40.4754	42.60075
484687.8	488999.5	499197.9	3577076	3514162	3757603	36.63231	35.74089	37.62147
476293.2	482462.5	495109.7	3092389	3025162	3258405	32.13639	31.09858	32.64936
464179.6	472624.3	485342.9	2616095	2542700	2763295	27.74512	26.56713	28.12688
446621.9	458270.3	471080.9	2151916	2070075	2277952	23.54869	22.1775	23.75508
420839.9	436973.6	449891.7	1705294	1611805	1806871	19.54106	17.91554	19.52549
385800.9	395681	414807.7	1284454	1174831	1356980	15.84401	13.85046	15.52296
338107.3	328456.5	360684.7	898653.1	779150.4	942171.9	12.26803	10.60792	12.00135
271873.5	241648.3	286315.6	560545.8	450693.9	581487.2	9.042334	7.779604	8.841444
186160.2	142070.1	191764.4	288672.3	209045.7	295171.6	6.173737	5.397995	6.05382
102512.1	66975.56	103407.1	102512.1	66975.56	103407.1	3.7	3.7	3.7

5.2. Reduction to 50 ug/m3												
FEMALES	SES: high, middle and low educational groups											
All Ages	Population	Cases	Rate	N		Mx-LSES	clean-l		Mx-MSES			Mx-HSES
25 to 29	523779	510	0.0010	5	1	0.001336	0.001336	1	0.000882	0.000882	1	5.64E-05
30 to 34	412827	620	0.0015	5	1.48	0.002061	0.001392	1.3	0.001361	0.001047	1.08	8.7E-05
35 to 39	340462	644	0.0019	5	1.48	0.002596	0.001754	1.3	0.001714	0.001318	1.08	0.00011
40 to 44	260845	758	0.0029	5	1.48	0.003988	0.002694	1.3	0.002633	0.002025	1.08	0.000168
45 to 49	198000	860	0.0043	5	1.48	0.00553	0.003737	1.3	0.003998	0.003075	1.08	0.003331
50 to 54	134158	898	0.0067	5	1.48	0.008522	0.005758	1.3	0.006161	0.004739	1.08	0.005134
55 to 59	109144	1074	0.0098	5	1.48	0.012529	0.008465	1.3	0.009057	0.006967	1.08	0.007547
60 to 64	82439	1297	0.0157	5	1.48	0.020031	0.013535	1.3	0.01448	0.011139	1.08	0.012067
65 to 69	57067	1440	0.0252	5	1.48	0.027556	0.018619	1.3	0.035353	0.027194	1.08	0.022774
70 to 74	37251	1545	0.0415	5	1.48	0.045294	0.030604	1.3	0.058108	0.044699	1.08	0.037433
75 to 79	22758	1588	0.0698	5	1.48	0.076201	0.051487	1.3	0.09776	0.0752	1.08	0.062976
80 to 84	14217	1812	0.1275	5	1.48	0.139186	0.094045	1.3	0.178565	0.137358	1.08	0.11503
85 and up	8605	2108	0.2450	3.7	1.48	0.267526	0.180761	1.3	0.343215	0.264012	1.08	0.221096

	Qx-LSES	Qx-MSES	Qx-HSES	Px-LSES	Px-MSES	Px-HSES	lx-LSES	lx-MSES	lx-HSES	dx-LSES	dx-MSES	dx-HSES
5.64E-05	0.006658	0.004401	0.000282	0.993342	0.995599	0.999718	100,000	100,000	100,000	665.8275	440.1351	28.18682
8.05E-05	0.006938	0.00522	0.000403	0.993062	0.99478	0.999597	99334.17	99559.86	99971.81	689.1904	519.697	40.2416
0.000101	0.008731	0.00657	0.000507	0.991269	0.99343	0.999493	98644.98	99040.17	99931.57	861.2285	650.6942	50.66071
0.000156	0.013381	0.010076	0.000779	0.986619	0.989924	0.999221	97783.75	98389.47	99880.91	1308.471	991.3322	77.77885
0.003085	0.01851	0.015258	0.015305	0.98149	0.984742	0.984695	96475.28	97398.14	99803.13	1785.734	1486.123	1527.483
0.004754	0.028383	0.023418	0.023489	0.971617	0.976582	0.976511	94689.55	95912.02	98275.65	2687.57	2246.025	2308.395
0.006988	0.041449	0.034238	0.034341	0.958551	0.965762	0.965659	92001.98	93665.99	95967.25	3813.396	3206.892	3295.648
0.011173	0.065458	0.054185	0.054347	0.934542	0.945815	0.945653	88188.58	90459.1	92671.61	5772.624	4901.49	5036.45
0.021087	0.088955	0.127317	0.100155	0.911045	0.872683	0.899845	82415.96	85557.61	87635.15	7331.342	10892.91	8777.089
0.03466	0.142143	0.201029	0.15948	0.857857	0.798971	0.84052	75084.62	74664.7	78858.07	10672.78	15009.76	12576.32
0.058311	0.228079	0.3165	0.254462	0.771921	0.6835	0.745538	64411.84	59654.94	66281.75	14690.97	18880.77	16866.16
0.106509	0.380713	0.511234	0.420561	0.619287	0.488766	0.579439	49720.87	40774.18	49415.58	18929.39	20845.16	20782.29
0.204718	0.501207	0.656295	0.549388	0.498793	0.343705	0.450612	30791.48	19929.01	28633.29	15432.9	13079.31	15730.79

Lx-LSES	Lx-MSES	Lx-HSES	Tx-LSES	Tx-MSES	Tx-HSES	Ex-LSES	Ex-MSES	Ex-HSES
498335.4	498899.7	499929.5	5134665	5048941	5270989	51.34665	50.48941	52.70989
494947.9	496500.1	499758.5	4636330	4550041	4771059	46.67407	45.70156	47.72405
491071.8	493574.1	499531.2	4141382	4053541	4271301	41.98269	40.92825	42.74226
485647.6	489469	499210.1	3650310	3559967	3771770	37.33043	36.1824	37.76267
477912.1	483275.4	495197	3164662	3070498	3272560	32.80283	31.52522	32.79015
466728.8	473945	485607.3	2686750	2587223	2777363	28.37431	26.97496	28.26094
450476.4	460312.7	471597.1	2220021	2113278	2291755	24.13015	22.56184	23.8806
426511.4	440041.8	450766.9	1769545	1652965	1820158	20.06547	18.27306	19.64095
393751.4	400555.8	416233.1	1343034	1212923	1369391	16.2958	14.17668	15.62605
348741.1	335799.1	362849.5	949282.2	812367.2	953158.2	12.64283	10.8802	12.08701
285331.8	251072.8	289243.3	600541.1	476568.1	590308.7	9.323459	7.988745	8.906053
201280.9	151758	195122.2	315209.3	225495.3	301065.4	6.339578	5.530346	6.092519
113928.5	73737.35	105943.2	113928.5	73737.35	105943.2	3.7	3.7	3.7

5.3. Reduction to background level												
FEMALES	SES: high, middle and low educational groups											
All Ages	Population	Cases	Rate	N		Mx-LSES	clean-l		Mx-MSES			Mx-HSES
25 to 29	523779	510	0.0010	5	1	0.001336	0.001336	1	0.000882	0.000882	1	5.64E-05
30 to 34	412827	620	0.0015	5	1.77	0.002061	0.001164	1.47	0.001361	0.000926	1.11	8.7E-05
35 to 39	340462	644	0.0019	5	1.77	0.002596	0.001466	1.47	0.001714	0.001166	1.11	0.00011
40 to 44	260845	758	0.0029	5	1.77	0.003988	0.002253	1.47	0.002633	0.001791	1.11	0.000168
45 to 49	198000	860	0.0043	5	1.77	0.00553	0.003124	1.47	0.003998	0.002719	1.11	0.003331
50 to 54	134158	898	0.0067	5	1.77	0.008522	0.004815	1.47	0.006161	0.004191	1.11	0.005134
55 to 59	109144	1074	0.0098	5	1.77	0.012529	0.007078	1.47	0.009057	0.006161	1.11	0.007547
60 to 64	82439	1297	0.0157	5	1.77	0.020031	0.011317	1.47	0.01448	0.009851	1.11	0.012067
65 to 69	57067	1440	0.0252	5	1.77	0.027556	0.015569	1.47	0.035353	0.02405	1.11	0.022774
70 to 74	37251	1545	0.0415	5	1.77	0.045294	0.02559	1.47	0.058108	0.039529	1.11	0.037433
75 to 79	22758	1588	0.0698	5	1.77	0.076201	0.043052	1.47	0.09776	0.066504	1.11	0.062976
80 to 84	14217	1812	0.1275	5	1.77	0.139186	0.078636	1.47	0.178565	0.121473	1.11	0.11503
85 and up	8605	2108	0.2450	3.7	1.77	0.267526	0.151144	1.47	0.343215	0.23348	1.11	0.221096

	Qx-LSES	Qx-MSES	Qx-HSES	Px-LSES	Px-MSES	Px-HSES	lx-LSES	lx-MSES	lx-HSES	dx-LSES	dx-MSES	dx-HSES
5.64E-05	0.006658	0.004401	0.000282	0.993342	0.995599	0.999718	100,000	100,000	100,000	665.8275	440.1351	28.18682
7.83E-05	0.005805	0.004618	0.000392	0.994195	0.995382	0.999608	99334.17	99559.86	99971.81	576.5999	459.7348	39.1542
9.87E-05	0.007305	0.005812	0.000493	0.992695	0.994188	0.999507	98757.57	99100.13	99932.66	721.4612	576.011	49.29238
0.000152	0.011201	0.008916	0.000758	0.988799	0.991084	0.999242	98036.11	98524.12	99883.37	1098.116	878.3999	75.67937
0.003001	0.015501	0.013506	0.014894	0.984499	0.986494	0.985106	96938	97645.72	99807.69	1502.596	1318.763	1486.575
0.004625	0.023788	0.020737	0.022861	0.976212	0.979263	0.977139	95435.4	96326.96	98321.11	2270.213	1997.579	2247.759
0.006799	0.034776	0.030338	0.033429	0.965224	0.969662	0.966571	93165.19	94329.38	96073.35	3239.918	2861.778	3211.612
0.010871	0.055028	0.048069	0.052917	0.944972	0.951931	0.947083	89925.27	91467.6	92861.74	4948.416	4396.752	4913.993
0.020517	0.074927	0.113428	0.09758	0.925073	0.886572	0.90242	84976.85	87070.85	87947.75	6367.042	9876.275	8581.947
0.033723	0.120255	0.179871	0.155505	0.879745	0.820129	0.844495	78609.81	77194.57	79365.8	9453.196	13885.1	12341.8
0.056735	0.194341	0.285116	0.248439	0.805659	0.714884	0.751561	69156.61	63309.47	67024	13439.97	18050.51	16651.35
0.10363	0.328584	0.465884	0.411534	0.671416	0.534116	0.588466	55716.65	45258.96	50372.65	18307.62	21085.43	20730.05
0.199185	0.437033	0.603291	0.538538	0.562967	0.396709	0.461462	37409.02	24173.53	29642.6	16348.96	14583.67	15963.67

Lx-LSES	Lx-MSES	Lx-HSES	Tx-LSES	Tx-MSES	Tx-HSES	Ex-LSES	Ex-MSES	Ex-HSES
498335.4	498899.7	499929.5	5282194	5148814	5291594	52.82194	51.48814	52.91594
495229.4	496650	499761.2	4783859	4649914	4791664	48.15924	46.70471	47.93015
491984.2	494060.6	499540.1	4288629	4153264	4291903	43.42583	41.90978	42.94795
487435.3	490424.6	499227.6	3796645	3659204	3792363	38.72701	37.14018	37.96791
480933.5	484931.7	495322	3309210	3168779	3293135	34.13739	32.4518	32.99481
471501.5	476640.8	485986.2	2828276	2683847	2797813	29.63551	27.86185	28.45588
457726.1	464492.4	472337.7	2356775	2207207	2311827	25.29673	23.39893	24.06315
437255.3	446346.1	452023.7	1899049	1742714	1839489	21.11808	19.0528	19.80891
408966.7	410663.5	418283.9	1461793	1296368	1387466	17.20225	14.88866	15.77602
369416.1	351260.1	365974.5	1052827	885704.5	969181.9	13.39307	11.47366	12.21158
312183.2	271421.1	293491.6	683410.7	534444.4	603207.4	9.882073	8.441775	8.999872
232814.2	173581.2	200038.1	371227.6	263023.3	309715.8	6.662776	5.811518	6.148491
138413.4	89442.05	109677.6	138413.4	89442.05	109677.6	3.7	3.7	3.7

Life Expectancy Tables 5: Comparison of Pollution Reduction Scenarios using relative risk coefficients of												
1.35, 1.23, and 1.06 respectively for low, middle and high educational levels												
5.1. Reduction to 60 ug/m3												
SES: high.middle and lower educational levels												
Males	Population	Deaths	DR		rr	Mx-LSES		clean-l	Mx-MSES		clean-M	Mx-HSES
All Ages						28%			49%			23%
25 to 29	487562	1414	0.0029	5	1	0.004175	1	0.004175	0.002799	1	0.002799	0.001564
30 to 34	397990	1380	0.0035	5	1.36	0.004992	1.23	0.00367	0.003346	1.06	0.002721	0.00187
35 to 39	337886	1458	0.0043	5	1.36	0.006212	1.23	0.004568	0.004165	1.06	0.003386	0.002327
40 to 44	260181	1539	0.0059	5	1.36	0.008515	1.23	0.006261	0.005709	1.06	0.004641	0.003189
45 to 49	200036	1854	0.0093	5	1.36	0.01188	1.23	0.008735	0.011198	1.06	0.009104	0.005894
50 to 54	129822	1763	0.0136	5	1.36	0.017407	1.23	0.012799	0.016407	1.06	0.013339	0.008635
55 to 59	98814	2074	0.0210	5	1.36	0.026903	1.23	0.019782	0.025359	1.06	0.020617	0.013347
60 to 64	70088	2204	0.0314	5	1.36	0.040307	1.23	0.029638	0.037993	1.06	0.030889	0.019996
65 to 69	45565	2172	0.0477	5	1.36	0.053754	1.23	0.039525	0.053754	1.06	0.043702	0.040115
70 to 74	26416	1951	0.0739	5	1.36	0.083285	1.23	0.061239	0.083285	1.06	0.067712	0.062153
75 to 79	14303	1594	0.1114	5	1.36	0.125672	1.23	0.092406	0.125672	1.06	0.102173	0.093785
80 to 84	7664	1302	0.1699	5	1.36	0.191573	1.23	0.140862	0.191573	1.06	0.15575	0.142965
85 and up	4,920	1165	0.2368	3.7	1.36	0.267017	1.23	0.196336	0.267017	1.06	0.217087	0.199267
	4,617,557											

clean-H	Qx-LSES	Qx-MSES	Qx-HSES	Px-LSES	Px-MSES	Px-HSES	lx-LSES	lx-MSES	lx-HSES	dx-LSES	dx-MSES	dx-HSES
0.001564	0.020659	0.013898	0.007788	0.979341	0.986102	0.992212	100,000	100,000	100,000	2065.94	1389.762	778.792
0.001764	0.018185	0.013512	0.00878	0.981815	0.986488	0.99122	97934.06	98610.24	99221.21	1780.912	1332.383	871.1465
0.002195	0.02258	0.016787	0.010914	0.97742	0.983213	0.989086	96153.15	97277.85	98350.06	2171.143	1632.995	1073.435
0.003009	0.030824	0.02294	0.014931	0.969176	0.97706	0.985069	93982	95644.86	97276.63	2896.883	2194.112	1452.471
0.00556	0.042743	0.044507	0.027419	0.957257	0.955493	0.972581	91085.12	93450.75	95824.15	3893.278	4159.21	2627.407
0.008147	0.062012	0.064544	0.03992	0.937988	0.935456	0.96008	87191.84	89291.54	93196.75	5406.916	5763.254	3720.441
0.012591	0.094249	0.098031	0.061035	0.905751	0.901969	0.938965	81784.93	83528.28	89476.31	7708.118	8188.37	5461.152
0.018864	0.137967	0.143372	0.090074	0.862033	0.856628	0.909926	74076.81	75339.91	84015.16	10220.13	10801.6	7567.596
0.037844	0.179852	0.196988	0.172865	0.820148	0.803012	0.827135	63856.68	64538.31	76447.56	11484.74	12713.3	13215.11
0.058635	0.265542	0.289545	0.255694	0.734458	0.710455	0.744306	52371.94	51825.01	63232.45	13906.97	15005.66	16168.17
0.088477	0.375325	0.406922	0.362256	0.624675	0.593078	0.637744	38464.97	36819.36	47064.28	14436.87	14982.62	17049.31
0.134873	0.520881	0.560505	0.504317	0.479119	0.439495	0.495683	24028.11	21836.73	30014.97	12515.78	12239.59	15137.05
0.187987	0.532888	0.573071	0.516075	0.467112	0.426929	0.483925	11512.33	9597.14	14877.93	6134.777	5499.843	7678.122

Lx-LSES	Lx-MSES	Lx-HSES	Tx-LSES	Tx-MSES	Tx-HSES	Ex-LSES	Ex-MSES	Ex-HSES
494835.2	496525.6	498053	4326025	4350316	4712841	43.26025	43.50316	47.12841
485218	489720.2	493928.2	3831189	3853791	4214788	39.12009	39.08104	42.4787
475337.9	482306.8	489066.7	3345971	3364071	3720860	34.79835	34.58208	37.83281
462667.8	472739	482752	2870633	2881764	3231793	30.5445	30.12984	33.22271
445692.4	456855.7	472552.3	2407966	2409025	2749041	26.43643	25.77855	28.68839
422441.9	432049.5	456682.6	1962273	1952169	2276489	22.50524	21.86287	24.4267
389654.3	397170.5	433728.7	1539831	1520120	1819806	18.82781	18.19886	20.33841
344833.7	349695.6	401156.8	1150177	1122949	1386077	15.52682	14.9051	16.49794
290571.6	290908.3	349200	805343.2	773253.6	984920.6	12.61173	11.98131	12.88361
227092.3	221610.9	275741.8	514771.7	482345.3	635720.5	9.82915	9.30719	10.05371
156232.7	146640.2	192698.1	287679.4	260734.3	359978.7	7.478996	7.081447	7.648661
88851.08	78584.68	112232.2	131446.7	114094.1	167280.6	5.470538	5.22487	5.573237
42595.6	35509.42	55048.32	42595.6	35509.42	55048.32	3.7	3.7	3.7

5.2. Reduction to 50 ug/m3												
SES: high.middle and lower educational levels												
Males	Population	Deaths	DR		rr	Mx-LSES		clean-l	Mx-MSES		clean-M	Mx-HSES
All Ages						28%			49%			23%
25 to 29	487562	1414	0.0029	5	1	0.004175	1	0.004175	0.002799	1	0.002799	0.001564
30 to 34	397990	1380	0.0035	5	1.48	0.004992	1.3	0.003373	0.003346	1.08	0.002574	0.00187
35 to 39	337886	1458	0.0043	5	1.48	0.006212	1.3	0.004197	0.004165	1.08	0.003203	0.002327
40 to 44	260181	1539	0.0059	5	1.48	0.008515	1.3	0.005754	0.005709	1.08	0.004391	0.003189
45 to 49	200036	1854	0.0093	5	1.48	0.01188	1.3	0.008027	0.011198	1.08	0.008614	0.005894
50 to 54	129822	1763	0.0136	5	1.48	0.017407	1.3	0.011761	0.016407	1.08	0.012621	0.008635
55 to 59	98814	2074	0.0210	5	1.48	0.026903	1.3	0.018178	0.025359	1.08	0.019507	0.013347
60 to 64	70088	2204	0.0314	5	1.48	0.040307	1.3	0.027235	0.037993	1.08	0.029225	0.019996
65 to 69	45565	2172	0.0477	5	1.48	0.053754	1.3	0.03632	0.053754	1.08	0.041349	0.040115
70 to 74	26416	1951	0.0739	5	1.48	0.083285	1.3	0.056274	0.083285	1.08	0.064066	0.062153
75 to 79	14303	1594	0.1114	5	1.48	0.125672	1.3	0.084914	0.125672	1.08	0.096671	0.093785
80 to 84	7664	1302	0.1699	5	1.48	0.191573	1.3	0.129441	0.191573	1.08	0.147364	0.142965
85 and up	4,920	1165	0.2368	3.7	1.48	0.267017	1.3	0.180417	0.267017	1.08	0.205398	0.199267

clean-H	Qx-LSES	Qx-MSES	Qx-HSES	Px-LSES	Px-MSES	Px-HSES	lx-LSES	lx-MSES	lx-HSES	dx-LSES	dx-MSES	dx-HSES
0.001564	0.020659	0.013898	0.007788	0.979341	0.986102	0.992212	100,000	100,000	100,000	2065.94	1389.762	778.792
0.001731	0.016723	0.012789	0.008618	0.983277	0.987211	0.991382	97934.06	98610.24	99221.21	1637.721	1261.098	855.0837
0.002154	0.020768	0.01589	0.010713	0.979232	0.98411	0.989287	96296.34	97349.14	98366.12	1999.906	1546.896	1053.836
0.002953	0.02836	0.021718	0.014657	0.97164	0.978282	0.985343	94296.43	95802.24	97312.29	2674.249	2080.668	1426.293
0.005457	0.039346	0.042161	0.026918	0.960654	0.957839	0.973082	91622.18	93721.57	95886	3604.948	3951.392	2581.07
0.007996	0.057127	0.061175	0.039196	0.942873	0.938825	0.960804	88017.24	89770.18	93304.93	5028.192	5491.699	3657.134
0.012358	0.086939	0.092998	0.059938	0.913061	0.907002	0.940062	82989.04	84278.48	89647.79	7214.988	7837.727	5373.329
0.018515	0.127493	0.136177	0.08848	0.872507	0.863823	0.91152	75774.06	76440.76	84274.46	9660.679	10409.49	7456.598
0.037143	0.166483	0.187375	0.169936	0.833517	0.812625	0.830064	66113.38	66031.27	76817.86	11006.76	12372.61	13054.11
0.057549	0.246667	0.276106	0.251555	0.753333	0.723894	0.748445	55106.61	53658.65	63763.76	13593	14815.48	16040.07
0.086838	0.350222	0.389276	0.356744	0.649778	0.610724	0.643256	41513.61	38843.17	47723.69	14538.99	15120.71	17025.13
0.132375	0.488973	0.538449	0.4973	0.511027	0.461551	0.5027	26974.63	23722.45	30698.55	13189.86	12773.34	15266.38
0.184506	0.500493	0.55071	0.50895	0.499507	0.44929	0.49105	13784.77	10949.12	15432.18	6899.179	6029.791	7854.203

Lx-LSES	Lx-MSES	Lx-HSES	Tx-LSES	Tx-MSES	Tx-HSES	Ex-LSES	Ex-MSES	Ex-HSES
494835.2	496525.6	498053	4418653	4409025	4730763	44.18653	44.09025	47.30763
485576	489898.4	493968.3	3923818	3912500	4232710	40.06592	39.67641	42.65932
476481.9	482878.5	489196	3438242	3422601	3738741	35.70481	35.158	38.00842
464796.5	473809.5	482995.7	2961760	2939723	3249545	31.40904	30.68532	33.39296
449098.5	458729.4	472977.3	2496964	2465913	2766550	27.25283	26.31105	28.85249
427515.7	435121.7	457381.8	2047865	2007184	2293572	23.26664	22.35914	24.58147
396907.7	401798.1	434805.6	1620350	1572062	1836191	19.52486	18.65319	20.48227
354718.6	356180.1	402730.8	1223442	1170264	1401385	16.14592	15.30943	16.62882
303050	299224.8	351454.1	868723.3	814084.1	998654.1	13.1399	12.32877	13.00028
241550.6	231254.6	278718.6	565673.3	514859.3	647200.1	10.26507	9.595084	10.14997
171220.6	156414.1	196055.6	324122.7	283604.7	368481.5	7.807625	7.301277	7.721144
101898.5	86678.93	115326.8	152902.1	127190.7	172425.9	5.668368	5.361615	5.616743
51003.63	40511.73	57099.05	51003.63	40511.73	57099.05	3.7	3.7	3.7

5.3. Reduction to background												
SES: high.middle and lower educational levels												
Males	Population	Deaths	DR		rr	Mx-LSES		clean-l	Mx-MSES		clean-M	Mx-HSES
All Ages						28%			49%			23%
25 to 29	487562	1414	0.0029	5	1	0.004175	1	0.004175	0.002799	1	0.002799	0.001564
30 to 34	397990	1380	0.0035	5	1.77	0.004992	1.47	0.00282	0.003346	1.11	0.002277	0.00187
35 to 39	337886	1458	0.0043	5	1.77	0.006212	1.47	0.00351	0.004165	1.11	0.002833	0.002327
40 to 44	260181	1539	0.0059	5	1.77	0.008515	1.47	0.004811	0.005709	1.11	0.003884	0.003189
45 to 49	200036	1854	0.0093	5	1.77	0.01188	1.47	0.006712	0.011198	1.11	0.007618	0.005894
50 to 54	129822	1763	0.0136	5	1.77	0.017407	1.47	0.009834	0.016407	1.11	0.011161	0.008635
55 to 59	98814	2074	0.0210	5	1.77	0.026903	1.47	0.0152	0.025359	1.11	0.017251	0.013347
60 to 64	70088	2204	0.0314	5	1.77	0.040307	1.47	0.022773	0.037993	1.11	0.025846	0.019996
65 to 69	45565	2172	0.0477	5	1.77	0.053754	1.47	0.030369	0.053754	1.11	0.036567	0.040115
70 to 74	26416	1951	0.0739	5	1.77	0.083285	1.47	0.047054	0.083285	1.11	0.056657	0.062153
75 to 79	14303	1594	0.1114	5	1.77	0.125672	1.47	0.071001	0.125672	1.11	0.085491	0.093785
80 to 84	7664	1302	0.1699	5	1.77	0.191573	1.47	0.108233	0.191573	1.11	0.130322	0.142965
85 and up	4,920	1165	0.2368	3.7	1.77	0.267017	1.47	0.150857	0.267017	1.11	0.181644	0.199267

clean-H	Qx-LSES	Qx-MSES	Qx-HSES	Px-LSES	Px-MSES	Px-HSES	lx-LSES	lx-MSES	lx-HSES	dx-LSES	dx-MSES	dx-HSES
0.001564	0.020659	0.013898	0.007788	0.979341	0.986102	0.992212	100,000	100,000	100,000	2065.94	1389.762	778.792
0.001684	0.014002	0.011318	0.008386	0.985998	0.988682	0.991614	97934.06	98610.24	99221.21	1371.273	1116.082	832.0702
0.002096	0.017395	0.014065	0.010425	0.982605	0.985935	0.989575	96562.79	97494.16	98389.14	1679.723	1371.301	1025.742
0.002873	0.023769	0.019231	0.014264	0.976231	0.980769	0.985736	94883.06	96122.85	97363.4	2255.246	1848.526	1388.749
0.00531	0.033006	0.037376	0.0262	0.966994	0.962624	0.9738	92627.82	94274.33	95974.65	3057.246	3523.628	2514.548
0.00778	0.047992	0.054292	0.038156	0.952008	0.945708	0.961844	89570.57	90750.7	93460.1	4298.681	4927.079	3566.099
0.012024	0.073216	0.082688	0.058366	0.926784	0.917312	0.941634	85271.89	85823.62	89894	6243.288	7096.561	5246.712
0.018015	0.10773	0.121385	0.086192	0.89227	0.878615	0.913808	79028.6	78727.06	84647.29	8513.726	9556.257	7295.888
0.036139	0.141131	0.167521	0.165724	0.858869	0.832479	0.834276	70514.88	69170.8	77351.4	9951.841	11587.55	12818.95
0.055994	0.210507	0.248137	0.245591	0.789493	0.751863	0.754409	60563.04	57583.25	64532.45	12748.92	14288.54	15848.58
0.084491	0.301491	0.352185	0.348784	0.698509	0.647815	0.651216	47814.12	43294.71	48683.88	14415.53	15247.75	16980.14
0.128797	0.42592	0.491482	0.487133	0.57408	0.508518	0.512867	33398.59	28046.96	31703.74	14225.12	13784.57	15443.93
0.17952	0.436383	0.503041	0.498624	0.563617	0.496959	0.501376	19173.47	14262.39	16259.81	8366.984	7174.572	8107.529

Lx-LSES	Lx-MSES	Lx-HSES	Tx-LSES	Tx-MSES	Tx-HSES	Ex-LSES	Ex-MSES	Ex-HSES
494835.2	496525.6	498053	4609723	4537920	4756917	46.09723	45.3792	47.56917
486242.1	490261	494025.9	4114887	4041395	4258864	42.01692	40.98352	42.92292
478614.6	484042.5	489381.3	3628645	3551134	3764838	37.57809	36.42407	38.26477
468777.2	475993	483345.1	3150031	3067091	3275457	33.19908	31.90803	33.64156
455496	462562.6	473586.9	2681254	2591098	2792112	28.94653	27.48466	29.09218
437106.2	441435.8	458385.2	2225758	2128536	2318525	24.84921	23.45476	24.80764
410751.2	411376.7	436353.2	1788651	1687100	1860140	20.97586	19.65776	20.69259
373858.7	369744.7	404996.7	1377900	1275723	1423786	17.43546	16.20438	16.82022
327694.8	316885.1	354709.6	1004041	905978.4	1018790	14.23872	13.0977	13.17093
270942.9	252194.9	283040.8	676346.6	589093.3	664080	11.16765	10.23029	10.29064
203031.8	178354.2	200969	405403.7	336898.4	381039.2	8.478746	7.781514	7.826805
131430.1	105773.4	119908.9	202372	158544.2	180070.2	6.059297	5.652813	5.679777
70941.84	52770.84	60161.3	70941.84	52770.84	60161.3	3.7	3.7	3.7

Life Expectancy Tables 6: Comparison of Pollution Reduction Scenarios using the general relative risk coefficient of the ACS Study: 1.17												
6.1. Reduction to 60 ug/m3												
SES: high.middle and lower educational levels												
Males	Population	Deaths	DR		rr	Mx-LSES		clean-l	Mx-MSES		clean-M	Mx-HSES
All Ages						28%			49%			23%
25 to 29	487562	1414	0.0029	5	1	0.004175	1	0.004175	0.002799	1	0.002799	0.001564
30 to 34	397990	1380	0.0035	5	1.17	0.004992	1.17	0.004266	0.003346	1.17	0.00286	0.00187
35 to 39	337886	1458	0.0043	5	1.17	0.006212	1.17	0.005309	0.004165	1.17	0.003559	0.002327
40 to 44	260181	1539	0.0059	5	1.17	0.008515	1.17	0.007278	0.005709	1.17	0.004879	0.003189
45 to 49	200036	1854	0.0093	5	1.17	0.01188	1.17	0.010154	0.011198	1.17	0.009571	0.005894
50 to 54	129822	1763	0.0136	5	1.17	0.017407	1.17	0.014878	0.016407	1.17	0.014023	0.008635
55 to 59	98814	2074	0.0210	5	1.17	0.026903	1.17	0.022994	0.025359	1.17	0.021674	0.013347
60 to 64	70088	2204	0.0314	5	1.17	0.040307	1.17	0.034451	0.037993	1.17	0.032473	0.019996
65 to 69	45565	2172	0.0477	5	1.17	0.053754	1.17	0.045943	0.053754	1.17	0.045943	0.040115
70 to 74	26416	1951	0.0739	5	1.17	0.083285	1.17	0.071184	0.083285	1.17	0.071184	0.062153
75 to 79	14303	1594	0.1114	5	1.17	0.125672	1.17	0.107412	0.125672	1.17	0.107412	0.093785
80 to 84	7664	1302	0.1699	5	1.17	0.191573	1.17	0.163738	0.191573	1.17	0.163738	0.142965
85 and up	4,920	1165	0.2368	3.7	1.17	0.267017	1.17	0.22822	0.267017	1.17	0.22822	0.199267
	4,617,557											

7/35 ug/m3 PM10 for 30 y/o and above for low, middle and high educational levels

clean-H	Qx-LSES	Qx-MSES	Qx-HSES	Px-LSES	Px-MSES	Px-HSES	lx-LSES	lx-MSES	lx-HSES	dx-LSES	dx-MSES	dx-HSES
0.001564	0.020659	0.013898	0.007788	0.979341	0.986102	0.992212	100,000	100,000	100,000	2065.94	1389.762	778.792
0.001598	0.021107	0.0142	0.007958	0.978893	0.9858	0.992042	97934.06	98610.24	99221.21	2067.068	1400.226	789.5697
0.001989	0.026199	0.01764	0.009893	0.973801	0.98236	0.990107	95866.99	97210.01	98431.64	2511.606	1714.803	973.8204
0.002726	0.03574	0.024102	0.013537	0.96426	0.975898	0.986463	93355.39	95495.21	97457.82	3336.515	2301.667	1319.291
0.005037	0.049513	0.046736	0.024873	0.950487	0.953264	0.975127	90018.87	93193.54	96138.53	4457.075	4355.498	2391.277
0.007381	0.071721	0.067742	0.036235	0.928279	0.932258	0.963765	85561.8	88838.04	93747.25	6136.567	6018.075	3396.94
0.011407	0.108722	0.1028	0.055455	0.891278	0.8972	0.944545	79425.23	82819.97	90350.31	8635.267	8513.888	5010.415
0.017091	0.158595	0.150172	0.081953	0.841405	0.849828	0.918047	70789.96	74306.08	85339.89	11226.92	11158.69	6993.832
0.034286	0.20605	0.20605	0.157896	0.79395	0.79395	0.842104	59563.04	63147.39	78346.06	12272.94	13011.49	12370.52
0.053122	0.30215	0.30215	0.234473	0.69785	0.69785	0.765527	47290.1	50135.9	65975.55	14288.7	15148.56	15469.48
0.080158	0.423373	0.423373	0.333883	0.576627	0.576627	0.666117	33001.4	34987.34	50506.07	13971.9	14812.69	16863.13
0.122192	0.5809	0.5809	0.467997	0.4191	0.4191	0.532003	19029.51	20174.65	33642.94	11054.24	11719.45	15744.8
0.170313	0.593735	0.593735	0.47918	0.406265	0.406265	0.52082	7975.266	8455.197	17898.14	4735.194	5020.146	8576.427

Lx-LSES	Lx-MSES	Lx-HSES	Tx-LSES	Tx-MSES	Tx-HSES	Ex-LSES	Ex-MSES	Ex-HSES
494835.2	496525.6	498053	4158628	4297014	4806755	41.58628	42.97014	48.06755
484502.6	489550.6	494132.1	3663793	3800489	4308702	37.41082	38.54051	43.42521
473055.9	481763.1	489723.6	3179291	3310938	3814570	33.16356	34.05964	38.75349
458435.6	471721.9	483990.9	2706235	2829175	3324846	28.98852	29.62635	34.11574
438951.7	455079	474714.4	2247799	2357453	2840855	24.97031	25.29631	29.5496
412467.6	429145	460243.9	1808847	1902374	2366141	21.14083	21.41396	25.23957
375538	392815.1	439225.5	1396380	1473229	1905897	17.58106	17.78833	21.09452
325882.5	343633.7	409214.9	1020842	1080414	1466671	14.42071	14.54005	17.18623
267132.9	283208.2	360804	694959.3	736780.2	1057456	11.66763	11.66763	13.49725
200728.8	212808.1	291204	427826.5	453571.9	696652.4	9.04685	9.04685	10.55925
130077.3	137905	210372.5	227097.7	240763.8	405448.3	6.881455	6.881455	8.027715
67511.93	71574.62	128852.7	97020.41	102858.9	195075.8	5.09842	5.09842	5.798418
29508.49	31284.23	66223.12	29508.49	31284.23	66223.12	3.7	3.7	3.7

6.2. Reduction to 50 ug/m3												
SES: high.middle and lower educational levels												
Males	Population	Deaths	DR		rr	Mx-LSES		clean-l	Mx-MSES		clean-M	Mx-HSES
All Ages						28%			49%			23%
25 to 29	487562	1414	0.0029	5	1	0.004175	1	0.004175	0.002799	1	0.002799	0.001564
30 to 34	397990	1380	0.0035	5	1.23	0.004992	1.23	0.004058	0.003346	1.23	0.002721	0.00187
35 to 39	337886	1458	0.0043	5	1.23	0.006212	1.23	0.00505	0.004165	1.23	0.003386	0.002327
40 to 44	260181	1539	0.0059	5	1.23	0.008515	1.23	0.006923	0.005709	1.23	0.004641	0.003189
45 to 49	200036	1854	0.0093	5	1.23	0.01188	1.23	0.009659	0.011198	1.23	0.009104	0.005894
50 to 54	129822	1763	0.0136	5	1.23	0.017407	1.23	0.014152	0.016407	1.23	0.013339	0.008635
55 to 59	98814	2074	0.0210	5	1.23	0.026903	1.23	0.021873	0.025359	1.23	0.020617	0.013347
60 to 64	70088	2204	0.0314	5	1.23	0.040307	1.23	0.03277	0.037993	1.23	0.030889	0.019996
65 to 69	45565	2172	0.0477	5	1.23	0.053754	1.23	0.043702	0.053754	1.23	0.043702	0.040115
70 to 74	26416	1951	0.0739	5	1.23	0.083285	1.23	0.067712	0.083285	1.23	0.067712	0.062153
75 to 79	14303	1594	0.1114	5	1.23	0.125672	1.23	0.102173	0.125672	1.23	0.102173	0.093785
80 to 84	7664	1302	0.1699	5	1.23	0.191573	1.23	0.15575	0.191573	1.23	0.15575	0.142965
85 and up	4,920	1165	0.2368	3.7	1.23	0.267017	1.23	0.217087	0.267017	1.23	0.217087	0.199267

clean-H	Qx-LSES	Qx-MSES	Qx-HSES	Px-LSES	Px-MSES	Px-HSES	Ix-LSES	Ix-MSES	Ix-HSES	dx-LSES	dx-MSES	dx-HSES
0.001564	0.020659	0.013898	0.007788	0.979341	0.986102	0.992212	100,000	100,000	100,000	2065.94	1389.762	778.792
0.00152	0.020087	0.013512	0.007571	0.979913	0.986488	0.992429	97934.06	98610.24	99221.21	1967.248	1332.383	751.1999
0.001892	0.024937	0.016787	0.009413	0.975063	0.983213	0.990587	95966.81	97277.85	98470.01	2393.106	1632.995	926.9017
0.002593	0.034026	0.02294	0.012881	0.965974	0.97706	0.987119	93573.71	95644.86	97543.11	3183.956	2194.112	1256.449
0.004792	0.047154	0.044507	0.023674	0.952846	0.955493	0.976326	90389.75	93450.75	96286.66	4262.271	4159.21	2279.517
0.007021	0.068342	0.064544	0.034498	0.931658	0.935456	0.965502	86127.48	89291.54	94007.14	5886.111	5763.254	3243.06
0.010851	0.103693	0.098031	0.052822	0.896307	0.901969	0.947178	80241.37	83528.28	90764.08	8320.502	8188.37	4794.316
0.016257	0.151444	0.143372	0.078111	0.848556	0.856628	0.921889	71920.87	75339.91	85969.77	10892	10801.6	6715.194
0.032613	0.196988	0.196988	0.150774	0.803012	0.803012	0.849226	61028.86	64538.31	79254.57	12021.98	12713.3	11949.55
0.050531	0.289545	0.289545	0.224318	0.710455	0.710455	0.775682	49006.89	51825.01	67305.02	14189.68	15005.66	15097.73
0.076248	0.406922	0.406922	0.320204	0.593078	0.593078	0.679796	34817.2	36819.36	52207.29	14167.9	14982.62	16716.98
0.116232	0.560505	0.560505	0.450308	0.439495	0.439495	0.549692	20649.3	21836.73	35490.32	11574.03	12239.59	15981.57
0.162005	0.573071	0.573071	0.461195	0.426929	0.426929	0.538805	9075.269	9597.14	19508.74	5200.774	5499.843	8997.34

Lx-LSES	Lx-MSES	Lx-HSES	Tx-LSES	Tx-MSES	Tx-HSES	Ex-LSES	Ex-MSES	Ex-HSES
494835.2	496525.6	498053	4214548	4350316	4853550	42.14548	43.50316	48.5355
484752.2	489720.2	494228	3719713	3853791	4355497	37.98181	39.08104	43.89684
473851.3	482306.8	490032.8	3234961	3364071	3861269	33.70916	34.58208	39.21264
459908.6	472739	484574.4	2761110	2881764	3371236	29.50732	30.12984	34.5615
441293.1	456855.7	475734.5	2301201	2409025	2886662	25.45865	25.77855	29.97987
415922.1	432049.5	461928.1	1859908	1952169	2410927	21.59482	21.86287	25.64621
380405.6	397170.5	441834.6	1443986	1520120	1948999	17.99553	18.19886	21.47324
332374.3	349695.6	413060.8	1063580	1122949	1507165	14.7882	14.9051	17.53133
275089.4	290908.3	366399	731205.8	773253.6	1094104	11.98131	11.98131	13.80493
209560.2	221610.9	298780.8	456116.4	482345.3	727704.8	9.30719	9.30719	10.81204
138666.3	146640.2	219244	246556.2	260734.3	428924	7.081447	7.081447	8.215787
74311.43	78584.68	137497.6	107889.9	114094.1	209680	5.22487	5.22487	5.90809
33578.5	35509.42	72182.35	33578.5	35509.42	72182.35	3.7	3.7	3.7

6.3. Reduction to background level												
SES: high.middle and lower educational levels												
Males	Population	Deaths	DR		rr	Mx-LSES		clean-l	Mx-MSES		clean-M	Mx-HSES
All Ages						28%			49%			23%
25 to 29	487562	1414	0.0029	5	1	0.004175	1	0.004175	0.002799	1	0.002799	0.001564
30 to 34	397990	1380	0.0035	5	1.35	0.004992	1.35	0.003698	0.003346	1.35	0.002479	0.00187
35 to 39	337886	1458	0.0043	5	1.35	0.006212	1.35	0.004601	0.004165	1.35	0.003085	0.002327
40 to 44	260181	1539	0.0059	5	1.35	0.008515	1.35	0.006308	0.005709	1.35	0.004229	0.003189
45 to 49	200036	1854	0.0093	5	1.35	0.01188	1.35	0.0088	0.011198	1.35	0.008295	0.005894
50 to 54	129822	1763	0.0136	5	1.35	0.017407	1.35	0.012894	0.016407	1.35	0.012154	0.008635
55 to 59	98814	2074	0.0210	5	1.35	0.026903	1.35	0.019928	0.025359	1.35	0.018784	0.013347
60 to 64	70088	2204	0.0314	5	1.35	0.040307	1.35	0.029857	0.037993	1.35	0.028143	0.019996
65 to 69	45565	2172	0.0477	5	1.35	0.053754	1.35	0.039817	0.053754	1.35	0.039817	0.040115
70 to 74	26416	1951	0.0739	5	1.35	0.083285	1.35	0.061693	0.083285	1.35	0.061693	0.062153
75 to 79	14303	1594	0.1114	5	1.35	0.125672	1.35	0.093091	0.125672	1.35	0.093091	0.093785
80 to 84	7664	1302	0.1699	5	1.35	0.191573	1.35	0.141906	0.191573	1.35	0.141906	0.142965
85 and up	4,920	1165	0.2368	3.7	1.35	0.267017	1.35	0.197791	0.267017	1.35	0.197791	0.199267

clean-H	Qx-LSES	Qx-MSES	Qx-HSES	Px-LSES	Px-MSES	Px-HSES	lx-LSES	lx-MSES	lx-HSES	dx-LSES	dx-MSES	dx-HSES
0.001564	0.020659	0.013898	0.007788	0.979341	0.986102	0.992212	100,000	100,000	100,000	2065.94	1389.762	778.792
0.001385	0.018318	0.012318	0.0069	0.981682	0.987682	0.9931	97934.06	98610.24	99221.21	1793.983	1214.679	684.657
0.001723	0.022745	0.015306	0.00858	0.977255	0.984694	0.99142	96140.08	97395.56	98536.55	2186.745	1490.753	845.4348
0.002362	0.031049	0.020922	0.011743	0.968951	0.979078	0.988257	93953.33	95904.81	97691.12	2917.118	2006.559	1147.158
0.004366	0.043053	0.040631	0.021593	0.956947	0.959369	0.978407	91036.21	93898.25	96543.96	3919.39	3815.196	2084.637
0.006397	0.062457	0.058976	0.03148	0.937543	0.941024	0.96852	87116.82	90083.05	94459.32	5441.031	5312.752	2973.56
0.009886	0.094914	0.089708	0.04824	0.905086	0.910292	0.95176	81675.79	84770.3	91485.76	7752.147	7604.581	4413.247
0.014812	0.138918	0.131465	0.071416	0.861082	0.868535	0.928584	73923.64	77165.72	87072.51	10269.29	10144.6	6218.356
0.029715	0.181064	0.181064	0.138299	0.818936	0.818936	0.861701	63654.35	67021.11	80854.16	11525.48	12135.08	11182.04
0.046039	0.267246	0.267246	0.206437	0.732754	0.732754	0.793563	52128.87	54886.04	69672.12	13931.26	14668.1	14382.89
0.069471	0.37758	0.37758	0.295953	0.62242	0.62242	0.704047	38197.61	40217.94	55289.23	14422.67	15185.5	16363.02
0.1059	0.523729	0.523729	0.41866	0.476271	0.476271	0.58134	23774.95	25032.44	38926.21	12451.63	13110.21	16296.83
0.147605	0.535778	0.535778	0.428993	0.464222	0.464222	0.571007	11323.32	11922.23	22629.38	6066.781	6387.661	9707.857

Lx-LSES	Lx-MSES	Lx-HSES	Tx-LSES	Tx-MSES	Tx-HSES	Ex-LSES	Ex-MSES	Ex-HSES
494835.2	496525.6	498053	4317883	4448845	4939063	43.17883	44.48845	49.39063
485185.3	490014.5	494394.4	3823048	3952319	4441010	39.03696	40.08021	44.75868
475233.5	483250.9	490569.2	3337863	3462305	3946616	34.71874	35.5489	40.0523
462473.9	474507.6	485587.7	2862629	2979054	3456046	30.46863	31.06261	35.37728
445382.6	459953.2	477508.2	2400155	2504546	2970459	26.36484	26.67298	30.76794
421981.5	437133.4	464862.7	1954773	2044593	2492951	22.43852	22.69676	26.39179
388998.6	404840	446395.7	1532791	1607460	2028088	18.76677	18.96253	22.16834
343945	360467.1	419816.7	1143793	1202620	1581692	15.47262	15.5849	18.16523
289458	304767.9	376315.7	799847.6	842152.6	1161875	12.56548	12.56548	14.37001
225816.2	237759.9	312403.4	510389.6	537384.8	785559.7	9.79092	9.79092	11.27509
154931.4	163125.9	235538.6	284573.4	299624.8	473156.3	7.45003	7.45003	8.557838
87745.67	92386.66	153889	129642	136498.9	237617.7	5.452881	5.452881	6.104311
41896.28	44112.24	83728.72	41896.28	44112.24	83728.72	3.7	3.7	3.7

Life Expectancy Tables 6: Comparison of Pollution Reduction Scenarios using the general relative risk coefficient of the ACS Study: 1.17												
6.1. Reduction to 60 ug/m3												
FEMALES	SES: high, middle and low educational groups											
All Ages	Population	Cases	Rate	N		Mx-LSES	clean-l		Mx-MSES			Mx-HSES
25 to 29	523779	510	0.0010	5	1	0.001336	0.001336	1	0.000882	0.000882	1	5.64E-05
30 to 34	412827	620	0.0015	5	1.17	0.002061	0.001761	1.17	0.001361	0.001163	1.17	8.7E-05
35 to 39	340462	644	0.0019	5	1.17	0.002596	0.002218	1.17	0.001714	0.001465	1.17	0.00011
40 to 44	260845	758	0.0029	5	1.17	0.003988	0.003408	1.17	0.002633	0.00225	1.17	0.000168
45 to 49	198000	860	0.0043	5	1.17	0.00553	0.004727	1.17	0.003998	0.003417	1.17	0.003331
50 to 54	134158	898	0.0067	5	1.17	0.008522	0.007284	1.17	0.006161	0.005266	1.17	0.005134
55 to 59	109144	1074	0.0098	5	1.17	0.012529	0.010708	1.17	0.009057	0.007741	1.17	0.007547
60 to 64	82439	1297	0.0157	5	1.17	0.020031	0.017121	1.17	0.01448	0.012376	1.17	0.012067
65 to 69	57067	1440	0.0252	5	1.17	0.027556	0.023553	1.17	0.035353	0.030216	1.17	0.022774
70 to 74	37251	1545	0.0415	5	1.17	0.045294	0.038712	1.17	0.058108	0.049665	1.17	0.037433
75 to 79	22758	1588	0.0698	5	1.17	0.076201	0.065129	1.17	0.09776	0.083556	1.17	0.062976
80 to 84	14217	1812	0.1275	5	1.17	0.139186	0.118962	1.17	0.178565	0.15262	1.17	0.11503
85 and up	8605	2108	0.2450	3.7	1.17	0.267526	0.228654	1.17	0.343215	0.293346	1.17	0.221096

7/35 ug/m3 PM10 for 30 y/o and above for low, middle and high educational levels

	Qx-LSES	Qx-MSES	Qx-HSES	Px-LSES	Px-MSES	Px-HSES	lx-LSES	lx-MSES	lx-HSES	dx-LSES	dx-MSES	dx-HSES
5.64E-05	0.006658	0.004401	0.000282	0.993342	0.995599	0.999718	100,000	100,000	100,000	665.8275	440.1351	28.18682
7.43E-05	0.008768	0.005798	0.000372	0.991232	0.994202	0.999628	99334.17	99559.86	99971.81	870.9958	577.2737	37.14666
9.36E-05	0.011031	0.007297	0.000468	0.988969	0.992703	0.999532	98463.18	98982.59	99934.67	1086.153	722.3095	46.7661
0.000144	0.016897	0.011189	0.000719	0.983103	0.988811	0.999281	97377.02	98260.28	99887.9	1645.358	1099.419	71.80304
0.002847	0.023357	0.016939	0.014136	0.976643	0.983061	0.985864	95731.67	97160.86	99816.1	2235.983	1645.83	1410.998
0.004388	0.035769	0.025986	0.021702	0.964231	0.974014	0.978298	93495.68	95515.03	98405.1	3344.223	2482.025	2135.563
0.006451	0.052145	0.037969	0.031742	0.947855	0.962031	0.968258	90151.46	93033.01	96269.54	4700.944	3532.414	3055.755
0.010314	0.082089	0.060024	0.050272	0.917911	0.939976	0.949728	85450.52	89500.59	93213.78	7014.578	5372.221	4686.025
0.019465	0.111214	0.140469	0.092808	0.888786	0.859531	0.907192	78435.94	84128.37	88527.76	8723.184	11817.46	8216.099
0.031994	0.176482	0.220898	0.148121	0.823518	0.779102	0.851879	69712.75	72310.91	80311.66	12303.04	15973.36	11895.87
0.053826	0.280048	0.34559	0.237209	0.719952	0.65441	0.762791	57409.71	56337.55	68415.79	16077.47	19469.67	16228.86
0.098316	0.458463	0.55235	0.394593	0.541537	0.44765	0.605407	41332.23	36867.88	52186.93	18949.29	20363.99	20592.61
0.188971	0.594529	0.703564	0.518074	0.405471	0.296436	0.481926	22382.95	16503.89	31594.32	13307.31	11611.54	16368.21

Lx-LSES	Lx-MSES	Lx-HSES	Tx-LSES	Tx-MSES	Tx-HSES	Ex-LSES	Ex-MSES	Ex-HSES
498335.4	498899.7	499929.5	4923246	4960609	5330590	49.23246	49.60609	53.3059
494493.4	496356.1	499766.2	4424910	4461709	4830660	44.5457	44.81434	48.32022
489600.5	493107.2	499556.4	3930417	3965353	4330894	39.91763	40.06112	43.33726
482771.7	488552.9	499260	3440817	3472246	3831338	35.33499	35.33723	38.35638
473068.4	481689.7	495553	2958045	2983693	3332078	30.89934	30.70879	33.38217
459117.9	471370.1	486686.6	2484977	2502003	2836525	26.57852	26.19486	28.82498
439004.9	456334	473708.3	2025859	2030633	2349838	22.47172	21.82702	24.40895
409716.1	434072.4	454353.8	1586854	1574299	1876130	18.57044	17.58982	20.12717
370371.7	391098.2	422098.5	1177138	1140227	1421776	15.00763	13.55341	16.06023
317806.2	321621.1	371818.6	806765.9	749128.5	999677.6	11.57272	10.35983	12.44748
246854.9	233013.6	301506.8	488959.7	427507.4	627858.9	8.517021	7.58832	9.177106
159288	133429.4	209453.1	242104.9	194493.8	326352.1	5.857531	5.275427	6.253522
82816.9	61064.39	116899	82816.9	61064.39	116899	3.7	3.7	3.7

6.2. Reduction to 50 ug/m3												
FEMALES	SES: high, middle and low educational groups											
All Ages	Population	Cases	Rate	N		Mx-LSES	clean-l		Mx-MSES			Mx-HSES
25 to 29	523779	510	0.0010	5	1	0.001336	0.001336	1	0.000882	0.000882	1	5.64E-05
30 to 34	412827	620	0.0015	5	1.23	0.002061	0.001675	1.23	0.001361	0.001106	1.23	8.7E-05
35 to 39	340462	644	0.0019	5	1.23	0.002596	0.00211	1.23	0.001714	0.001393	1.23	0.00011
40 to 44	260845	758	0.0029	5	1.23	0.003988	0.003242	1.23	0.002633	0.002141	1.23	0.000168
45 to 49	198000	860	0.0043	5	1.23	0.00553	0.004496	1.23	0.003998	0.00325	1.23	0.003331
50 to 54	134158	898	0.0067	5	1.23	0.008522	0.006929	1.23	0.006161	0.005009	1.23	0.005134
55 to 59	109144	1074	0.0098	5	1.23	0.012529	0.010186	1.23	0.009057	0.007363	1.23	0.007547
60 to 64	82439	1297	0.0157	5	1.23	0.020031	0.016285	1.23	0.01448	0.011773	1.23	0.012067
65 to 69	57067	1440	0.0252	5	1.23	0.027556	0.022404	1.23	0.035353	0.028742	1.23	0.022774
70 to 74	37251	1545	0.0415	5	1.23	0.045294	0.036824	1.23	0.058108	0.047242	1.23	0.037433
75 to 79	22758	1588	0.0698	5	1.23	0.076201	0.061952	1.23	0.09776	0.07948	1.23	0.062976
80 to 84	14217	1812	0.1275	5	1.23	0.139186	0.113159	1.23	0.178565	0.145175	1.23	0.11503
85 and up	8605	2108	0.2450	3.7	1.23	0.267526	0.2175	1.23	0.343215	0.279037	1.23	0.221096

	Qx-LSES	Qx-MSES	Qx-HSES	Px-LSES	Px-MSES	Px-HSES	lx-LSES	lx-MSES	lx-HSES	dx-LSES	dx-MSES	dx-HSES
5.64E-05	0.006658	0.004401	0.000282	0.993342	0.995599	0.999718	100,000	100,000	100,000	665.8275	440.1351	28.18682
7.07E-05	0.008342	0.005516	0.000353	0.991658	0.994484	0.999647	99334.17	99559.86	99971.81	828.6855	549.1917	35.33495
8.9E-05	0.010496	0.006943	0.000445	0.989504	0.993057	0.999555	98505.49	99010.67	99936.48	1033.892	687.3922	44.48614
0.000137	0.016079	0.010646	0.000684	0.983921	0.989354	0.999316	97471.59	98323.28	99891.99	1567.263	1046.745	68.30444
0.002708	0.02223	0.01612	0.013451	0.97777	0.98388	0.986549	95904.33	97276.54	99823.69	2131.961	1568.058	1342.734
0.004174	0.034054	0.024734	0.020654	0.965946	0.975266	0.979346	93772.37	95708.48	98480.95	3193.29	2367.233	2034.031
0.006136	0.049664	0.036151	0.030217	0.950336	0.963849	0.969783	90579.08	93341.25	96446.92	4498.562	3374.359	2914.306
0.009811	0.078242	0.05718	0.047878	0.921758	0.94282	0.952122	86080.52	89966.89	93532.62	6735.083	5144.316	4478.176
0.018515	0.106077	0.134077	0.088481	0.893923	0.865923	0.911519	79345.44	84822.57	89054.44	8416.708	11372.72	7879.646
0.030433	0.168599	0.211261	0.141407	0.831401	0.788739	0.858593	70928.73	73449.85	81174.79	11958.5	15517.09	11478.66
0.0512	0.268219	0.331526	0.226951	0.731781	0.668474	0.773049	58970.23	57932.76	69696.13	15816.95	19206.22	15817.61
0.09352	0.44103	0.532581	0.378992	0.55897	0.467419	0.621008	43153.28	38726.54	53878.52	19031.9	20625.04	20419.55
0.179752	0.573849	0.680929	0.499109	0.426151	0.319071	0.500891	24121.38	18101.5	33458.97	13842.02	12325.83	16699.68

Lx-LSES	Lx-MSES	Lx-HSES	Tx-LSES	Tx-MSES	Tx-HSES	Ex-LSES	Ex-MSES	Ex-HSES
498335.4	498899.7	499929.5	4969779	5002823	5366887	49.69779	50.02823	53.66887
494599.1	496426.3	499770.7	4471443	4503923	4866958	45.01415	45.23834	48.6833
489942.7	493334.9	499571.2	3976844	4007497	4367187	40.3718	40.4754	43.69963
483439.8	488999.5	499289.2	3486901	3514162	3867616	35.77351	35.74089	38.71798
474191.8	482462.5	495761.6	3003462	3025162	3368327	31.31727	31.09858	33.74276
460878.6	472624.3	487319.7	2529270	2542700	2872565	26.97244	26.56713	29.16874
441649	458270.3	474948.8	2068391	2070075	2385245	22.8352	22.1775	24.73117
413564.9	436973.6	456467.6	1626742	1611805	1910297	18.89791	17.91554	20.42386
375685.4	395681	425573.1	1213177	1174831	1453829	15.28982	13.85046	16.32517
324747.4	328456.5	377177.3	837491.9	779150.4	1028256	11.80751	10.60792	12.66718
255308.8	241648.3	308936.6	512744.5	450693.9	651078.6	8.694973	7.779604	9.341674
168186.6	142070.1	218343.7	257435.7	209045.7	342141.9	5.965612	5.397995	6.350247
89249.1	66975.56	123798.2	89249.1	66975.56	123798.2	3.7	3.7	3.7

6.3. Reduction to background level												
FEMALES	SES: high, middle and low educational groups											
All Ages	Population	Cases	Rate	N		Mx-LSES	clean-l		Mx-MSES			Mx-HSES
25 to 29	523779	510	0.0010	5	1	0.001336	0.001336	1	0.000882	0.000882	1	5.64E-05
30 to 34	412827	620	0.0015	5	1.35	0.002061	0.001527	1.35	0.001361	0.001008	1.35	8.7E-05
35 to 39	340462	644	0.0019	5	1.35	0.002596	0.001923	1.35	0.001714	0.00127	1.35	0.00011
40 to 44	260845	758	0.0029	5	1.35	0.003988	0.002954	1.35	0.002633	0.00195	1.35	0.000168
45 to 49	198000	860	0.0043	5	1.35	0.00553	0.004096	1.35	0.003998	0.002961	1.35	0.003331
50 to 54	134158	898	0.0067	5	1.35	0.008522	0.006313	1.35	0.006161	0.004563	1.35	0.005134
55 to 59	109144	1074	0.0098	5	1.35	0.012529	0.00928	1.35	0.009057	0.006709	1.35	0.007547
60 to 64	82439	1297	0.0157	5	1.35	0.020031	0.014838	1.35	0.01448	0.010726	1.35	0.012067
65 to 69	57067	1440	0.0252	5	1.35	0.027556	0.020412	1.35	0.035353	0.026187	1.35	0.022774
70 to 74	37251	1545	0.0415	5	1.35	0.045294	0.033551	1.35	0.058108	0.043043	1.35	0.037433
75 to 79	22758	1588	0.0698	5	1.35	0.076201	0.056445	1.35	0.09776	0.072415	1.35	0.062976
80 to 84	14217	1812	0.1275	5	1.35	0.139186	0.103101	1.35	0.178565	0.132271	1.35	0.11503
85 and up	8605	2108	0.2450	3.7	1.35	0.267526	0.198167	1.35	0.343215	0.254234	1.35	0.221096

	Qx-LSES	Qx-MSES	Qx-HSES	Px-LSES	Px-MSES	Px-HSES	lx-LSES	lx-MSES	lx-HSES	dx-LSES	dx-MSES	dx-HSES
5.64E-05	0.006658	0.004401	0.000282	0.993342	0.995599	0.999718	100,000	100,000	100,000	665.8275	440.1351	28.18682
6.44E-05	0.007604	0.005027	0.000322	0.992396	0.994973	0.999678	99334.17	99559.86	99971.81	755.3046	500.4974	32.19457
8.11E-05	0.009567	0.006327	0.000406	0.990433	0.993673	0.999594	98578.87	99059.37	99939.62	943.1324	626.7921	40.53389
0.000125	0.01466	0.009704	0.000623	0.98534	0.990296	0.999377	97635.74	98432.58	99899.08	1431.378	955.2127	62.23925
0.002468	0.020274	0.014697	0.012263	0.979726	0.985303	0.987737	96204.36	97477.36	99836.85	1950.457	1432.651	1224.273
0.003803	0.031074	0.02256	0.018835	0.968926	0.97744	0.981165	94253.9	96044.71	98612.57	2928.815	2166.771	1857.41
0.005591	0.04535	0.03299	0.027568	0.95465	0.96701	0.972432	91325.09	93877.94	96755.16	4141.588	3097.07	2667.325
0.008938	0.071536	0.05223	0.043715	0.928464	0.94777	0.956285	87183.5	90780.87	94087.84	6236.724	4741.5	4113.088
0.01687	0.097106	0.122891	0.080935	0.902894	0.877109	0.919065	80946.77	86039.37	89974.75	7860.38	10573.46	7282.062
0.027728	0.154772	0.194307	0.129652	0.845228	0.805693	0.870348	73086.39	75465.91	82692.69	11311.73	14663.53	10721.28
0.046649	0.247326	0.306574	0.208885	0.752674	0.693426	0.791115	61774.67	60802.38	71971.41	15278.47	18640.44	15033.72
0.085207	0.409861	0.497005	0.35122	0.590139	0.502995	0.64878	46496.2	42161.93	56937.69	19057	20954.7	19997.66
0.163774	0.536524	0.639763	0.46506	0.463476	0.360237	0.53494	27439.2	21207.24	36940.03	14721.78	13567.61	17179.34

Lx-LSES	Lx-MSES	Lx-HSES	Tx-LSES	Tx-MSES	Tx-HSES	Ex-LSES	Ex-MSES	Ex-HSES
498335.4	498899.7	499929.5	5054221	5079996	5432426	50.54221	50.79996	54.32426
494782.6	496548.1	499778.6	4555886	4581097	4932496	45.86423	46.01349	49.33887
490536.5	493729.9	499596.8	4061103	4084549	4432717	41.19649	41.23334	44.35396
484600.2	489774.8	499339.8	3570567	3590819	3933121	36.57029	36.47998	39.37094
476145.6	483805.2	496123.5	3085966	3101044	3433781	32.0772	31.81296	34.39392
463947.5	474806.6	488419.3	2609821	2617239	2937657	27.68926	27.25021	29.78989
446271.5	461647	477107.5	2145873	2142432	2449238	23.49709	22.82146	25.31377
420325.7	442050.6	460156.5	1699602	1680785	1972130	19.49454	18.51475	20.96052
385082.9	403763.2	431668.6	1279276	1238734	1511974	15.80392	14.3973	16.80443
337152.6	340670.7	386660.2	894193.3	834971.2	1080305	12.23474	11.06422	13.0641
270677.2	257410.8	322272.7	557040.7	494300.5	693645.1	9.0173	8.129624	9.637787
184838.5	158422.9	234694.3	286363.5	236889.7	371372.4	6.158859	5.618568	6.522435
101525	78466.78	136678.1	101525	78466.78	136678.1	3.7	3.7	3.7

	A	B	C	D	E	F	G	H	I	J	K	L	M
1	Cause of Death Elimination Life Expectancy Table					Method 1							
2	SEX/AGE	n(no.of intervals)	male populati on MD	C-P deaths	Other Deaths	Total deaths	Mx-total	Mx-CP	Mx-others	RR-CP	MxCPclean	Mxtotalclean	QxTotal
3	<1	1	140,415	748	2886	3,634	0.0259	0.0053	0.0206	1.91	0.0028	0.0233	0.0231
4	1 YR-4YR	4	487,282	468	642	1,110	0.0023	0.0010	0.0013	1.00	0.0010	0.0023	0.0091
5	5yr-9yr	5	509576	128	293	421	0.0008	0.0003	0.0006	1.00	0.0003	0.0008	0.0041
6	10yr-14	5	438531	83	228	311	0.0007	0.0002	0.0005	1.00	0.0002	0.0007	0.0035
7	15yr-19	5	463906	151	419	570	0.0012	0.0003	0.0009	1.00	0.0003	0.0012	0.0061
8	20yr-24	5	496600	311	869	1,180	0.0024	0.0006	0.0017	1.00	0.0006	0.0024	0.0118
9	25yr-29	5	487562	470	944	1,414	0.0029	0.0010	0.0019	1.00	0.0010	0.0029	0.0144
10	30yr-34	5	397990	523	857	1,380	0.0035	0.0013	0.0022	1.36	0.0010	0.0031	0.0155
11	35yr-39	5	337886	683	775	1,458	0.0043	0.0020	0.0023	1.36	0.0015	0.0038	0.0187
12	40yr-44	5	260181	826	713	1,539	0.0059	0.0032	0.0027	1.36	0.0023	0.0051	0.0251
13	45yr-49	5	200036	1,119	735	1,854	0.0093	0.0056	0.0037	1.36	0.0041	0.0078	0.0382
14	50yr-54	5	129822	1,126	637	1,763	0.0136	0.0087	0.0049	1.36	0.0064	0.0113	0.0549
15	55yr-59	5	98814	1,430	644	2,074	0.0210	0.0145	0.0065	1.36	0.0106	0.0172	0.0823
16	60yr-64	5	70088	1,532	672	2,204	0.0314	0.0219	0.0096	1.36	0.0161	0.0257	0.1206
17	65yr-69	5	45565	1,536	636	2,172	0.0477	0.0337	0.0140	1.36	0.0248	0.0387	0.1766
18	70yr-74	5	26416	1,431	520	1,951	0.0739	0.0542	0.0197	1.36	0.0398	0.0595	0.2590
19	75yr-79	5	14303	1,167	427	1,594	0.1114	0.0816	0.0299	1.36	0.0600	0.0898	0.3668
20	80yr-84	5	7664	964	338	1,302	0.1699	0.1258	0.0441	1.36	0.0925	0.1366	0.5091
21	85&over	3.7	4,920	876	289	1,165	0.2368	0.1780	0.0587	1.36	0.1309	0.1897	0.5195
22	total		########	15,572	13524	29,096	0.0063						
23				*RR for C-P = 1.09 from the Pooled Estimate of the Pope and Dockery studiesfor 30 yrs and older								H3-(H3*((J3-1)/J3))	
24				*RR for Pulmonary causes = 1.2 from the Woodruff Study for less than 1 year old								K3+I3	
25													
26	Cause of Death Elimination Life Expectancy Table					Method 2							
27	SEX/AGE	n(no.of intervals)	male populati on MD	C-P deaths	Other deaths	Total deaths	Mx-total	Mx-CP	Mx-others	RR-CP	MxCPclean	Mxtotalclean	QxTotal
28	<1	1	140,415	748	2886	3634	0.02588	0.00533	0.02055	3.35	0.00159	0.02214	0.02195
29	1 YR-4YR	4	487,282	468	642	1110	0.00228	0.00096	0.00132	1.00	0.00096	0.00228	0.00908
30	5yr-9yr	5	509576	128	293	421	0.00083	0.00025	0.00057	1.00	0.00025	0.00083	0.00412
31	10yr-14	5	438531	83	228	311	0.00071	0.00019	0.00052	1.00	0.00019	0.00071	0.00354
32	15yr-19	5	463906	151	419	570	0.00123	0.00033	0.00090	1.00	0.00033	0.00123	0.00612
33	20yr-24	5	496600	311	869	1180	0.00238	0.00063	0.00175	1.00	0.00063	0.00238	0.01181
34	25yr-29	5	487562	470	944	1414	0.00290	0.00096	0.00194	1.00	0.00096	0.00290	0.01440
35	30yr-34	5	397990	523	857	1380	0.00347	0.00131	0.00215	1.77	0.00074	0.00290	0.01437
36	35yr-39	5	337886	683	775	1458	0.00432	0.00202	0.00229	1.77	0.00114	0.00344	0.01703
37	40yr-44	5	260181	826	713	1539	0.00592	0.00317	0.00274	1.77	0.00179	0.00453	0.02242

	A	B	C	D	E	F	G	H	I	J	K	L	M
38	45yr-49	5	200036	1,119	735	1854	0.00927	0.00559	0.00367	1.77	0.00316	0.00683	0.03360
39	50yr-54	5	129822	1,126	637	1763	0.01358	0.00867	0.00491	1.77	0.00490	0.00981	0.04786
40	55yr-59	5	98814	1,430	644	2074	0.02099	0.01447	0.00652	1.77	0.00818	0.01469	0.07086
41	60yr-64	5	70088	1,532	672	2204	0.03145	0.02186	0.00959	1.77	0.01235	0.02194	0.10398
42	65yr-69	5	45565	1,536	636	2172	0.04767	0.03371	0.01396	1.77	0.01905	0.03300	0.15244
43	70yr-74	5	26416	1,431	520	1951	0.07386	0.05417	0.01969	1.77	0.03061	0.05029	0.22337
44	75yr-79	5	14303	1,167	427	1594	0.11145	0.08159	0.02985	1.77	0.04610	0.07595	0.31915
45	80yr-84	5	7664	964	338	1302	0.16989	0.12578	0.04410	1.77	0.07106	0.11517	0.44710
46	85&over	3.7	4,920	876	289	1165	0.23679	0.17805	0.05874	1.77	0.10059	0.15933	0.45532
47			########	15572									
48													
49													
50													
51	Cause of Death Elimination Life Expectancy Table					Method 3							
52	SEX/AGE	n(no.of intervals)	male population MD	C-P deaths	Other deaths	Total deaths	Mx-total	Mx-CP	Mx-others	RR-CP	MxCPclean	Mxtotalclean	QxTotal
53	<1	1	140,415	748	2886	3634	0.02588	0.00533	0.02055	2.30	0.00232	0.02287	0.02266
54	1 YR-4YR	4	487,282	468	642	1110	0.00228	0.00096	0.00132	1.00	0.00096	0.00228	0.00908
55	5yr-9yr	5	509576	128	293	421	0.00083	0.00025	0.00057	1.00	0.00025	0.00083	0.00412
56	10yr-14	5	438531	83	228	311	0.00071	0.00019	0.00052	1.00	0.00019	0.00071	0.00354
57	15yr-19	5	463906	151	419	570	0.00123	0.00033	0.00090	1.00	0.00033	0.00123	0.00612
58	20yr-24	5	496600	311	869	1180	0.00238	0.00063	0.00175	1.00	0.00063	0.00238	0.01181
59	25yr-29	5	487562	470	944	1414	0.00290	0.00096	0.00194	1.00	0.00096	0.00290	0.01440
60	30yr-34	5	397990	523	857	1380	0.00347	0.00131	0.00215	1.48	0.00089	0.00304	0.01509
61	35yr-39	5	337886	683	775	1458	0.00432	0.00202	0.00229	1.48	0.00137	0.00366	0.01813
62	40yr-44	5	260181	826	713	1539	0.00592	0.00317	0.00274	1.48	0.00215	0.00489	0.02413
63	45yr-49	5	200036	1,119	735	1854	0.00927	0.00559	0.00367	1.48	0.00378	0.00745	0.03659
64	50yr-54	5	129822	1,126	637	1763	0.01358	0.00867	0.00491	1.48	0.00586	0.01077	0.05242
65	55yr-59	5	98814	1,430	644	2074	0.02099	0.01447	0.00652	1.48	0.00978	0.01630	0.07829
66	60yr-64	5	70088	1,532	672	2204	0.03145	0.02186	0.00959	1.48	0.01477	0.02436	0.11479
67	65yr-69	5	45565	1,536	636	2172	0.04767	0.03371	0.01396	1.48	0.02278	0.03674	0.16823
68	70yr-74	5	26416	1,431	520	1951	0.07386	0.05417	0.01969	1.48	0.03660	0.05629	0.24672
69	75yr-79	5	14303	1,167	427	1594	0.11145	0.08159	0.02985	1.48	0.05513	0.08498	0.35046
70	80yr-84	5	7664	964	338	1302	0.16989	0.12578	0.04410	1.48	0.08499	0.12909	0.48797
71	85&over	3.7	4,920	876	289	1165	0.23679	0.17805	0.05874	1.48	0.12030	0.17904	0.49763
72			########	15572									
73													

	N	O	P	Q	R	S
1						
2	Px	lxTotal	dxtotal	Lx	Txtotal	Extotal
3	0.9769	100,000	2312.65	98612.41	6616425.75	66.16
4	0.9909	97687.35	886.87	388620.92	6517813.34	66.72
5	0.9959	96800.48	399.05	483004.79	6129192.42	63.32
6	0.9965	96401.43	341.23	481154.10	5646187.64	58.57
7	0.9939	96060.21	588.34	478830.19	5165033.54	53.77
8	0.9882	95471.87	1127.58	474540.39	4686203.35	49.08
9	0.9856	94344.29	1358.21	468325.90	4211662.96	44.64
10	0.9845	92986.07	1439.16	461332.47	3743337.07	40.26
11	0.9813	91546.91	1714.03	453449.48	3282004.60	35.85
12	0.9749	89832.88	2250.84	443537.29	2828555.12	31.49
13	0.9618	87582.04	3345.13	429547.37	2385017.83	27.23
14	0.9451	84236.91	4622.34	409628.68	1955470.46	23.21
15	0.9177	79614.56	6549.28	381699.63	1545841.78	19.42
16	0.8794	73065.29	8809.23	343303.36	1164142.15	15.93
17	0.8234	64256.06	11348.71	292908.52	820838.79	12.77
18	0.7410	52907.35	13705.24	230273.64	527930.28	9.98
19	0.6332	39202.11	14380.84	160058.43	297656.64	7.59
20	0.4909	24821.26	12636.58	92514.87	137598.21	5.54
21	0.4805	12184.68	6329.58	45083.33	45083.33	3.70
22						
23	(B3*L3)/(1+(B3*(0.4*L3)))			O3*N3	B3*(O4+(0.4*P3))	
24	1-M3			O3*M3		
25						
26						
27	Px	lxTotal	dxtotal	Lx	Txtotal	Extotal
28	0.97805	100,000	2194.91	98663.05	6769975.10	67.70
29	0.99092	97805.09	887.94	389089.30	6671292.05	68.21
30	0.99588	96917.15	399.53	483586.92	6282202.75	64.82
31	0.99646	96517.62	341.64	481734.00	5798615.83	60.08
32	0.99388	96175.98	589.05	479407.28	5316881.84	55.28
33	0.98819	95586.93	1128.94	475112.31	4837474.55	50.61
34	0.98560	94457.99	1359.85	468890.34	4362362.24	46.18
35	0.98563	93098.14	1338.26	462145.07	3893471.90	41.82
36	0.98297	91759.88	1562.87	454892.24	3431326.83	37.39
37	0.97758	90197.01	2021.86	445930.41	2976434.59	33.00

	N	O	P	Q	R	S
38	0.95540	88175.15	2962.67	433469.09	2530504.19	28.70
39	0.95214	85212.48	4078.38	415866.46	2097035.10	24.61
40	0.92914	81134.10	5749.47	391296.84	1681168.64	20.72
41	0.89602	75384.64	7838.75	357326.30	1289871.80	17.11
42	0.84756	67545.89	10296.64	311987.84	932545.50	13.81
43	0.77663	57249.25	12787.72	254276.94	620557.66	10.84
44	0.68085	44461.53	14190.05	185832.51	366280.72	8.24
45	0.55290	30271.48	13534.46	117521.24	179448.21	5.93
46	0.54468	16737.02	7620.67	61926.97	61926.97	3.70
47						
48						
49						
50						
51						
52	Px	lxTotal	dxtotal	Lx	Txtotal	Extotal
53	0.97734	100,000	2266.22	98540.27	6668284.27	66.68
54	0.99092	97733.78	887.29	388805.63	6559644.00	67.22
55	0.99588	96846.49	399.24	483234.36	6180838.37	63.82
56	0.99646	96447.25	341.39	481382.79	5697604.02	59.07
57	0.99388	96105.86	588.62	479057.77	5216221.23	54.28
58	0.98819	95517.25	1128.12	474765.93	4737163.46	49.59
59	0.98560	94389.13	1356.86	468548.49	4262397.52	45.16
60	0.98491	93030.27	1403.96	461641.45	3793849.03	40.78
61	0.98167	91626.31	1661.32	453978.25	3332207.58	36.37
62	0.97587	89964.99	2171.09	444397.21	2878229.33	31.99
63	0.96341	87793.90	3212.25	430938.87	2433832.12	27.72
64	0.94758	84581.65	4434.15	411822.87	2002893.25	23.68
65	0.92171	80147.50	6274.57	385051.07	1591070.38	19.85
66	0.88521	73872.93	8480.24	348164.04	1206019.31	16.33
67	0.83177	65392.69	11000.77	299461.52	857855.27	13.12
68	0.75328	54391.92	13419.55	238410.71	558393.76	10.27
69	0.64954	40972.36	14359.09	168964.09	319983.05	7.81
70	0.51203	26613.27	12986.53	100600.02	151018.96	5.67
71	0.50237	13626.74	6781.07	50418.94	50418.94	3.70
72						
73						

Cause of Death Elimination Life Expectancy Table — Method 1

SEX/AGE	n(no.of Intervals)	Female Population	C-P Deaths	Other Deat	ToTal death	MxTotal	Mx-CP	Mx-others	RR-CP	xCPclean	xtotalclea	QxTotal	lxTotal	dxtotal	Lx	Txtotal	Extotal
<1	1	129201	528	2,020	2,548	0.019721	0.004057	0.015635	1.91	0.00214	0.017774	0.017649	100000	1764.869	98941.08	7346663	73.46563
1 YR-4YR	4	455632	434	443	877	0.001925	0.000953	0.000972	1	0.000953	0.001925	0.007676	98235.13	754.0094	391130.9	7247722	73.77933
5yr-9yr	5	478433	118	190	308	0.000644	0.000247	0.000397	1	0.000247	0.000644	0.003214	97481.12	313.2721	486622.4	6856591	70.33763
10yr-14	5	433907	110	151	261	0.000602	0.000254	0.000348	1	0.000254	0.000602	0.003003	97167.85	291.799	485109.8	6369969	65.55634
15yr-19	5	556091	113	191	304	0.000547	0.000203	0.000343	1	0.000203	0.000547	0.00273	96876.05	264.4363	483719.2	5884859	60.74627
20yr-24	5	581667	201	229	430	0.000739	0.000346	0.000394	1	0.000346	0.000739	0.003689	96611.61	356.4441	482167	5401140	55.9057
25yr-29	5	523779	221	289	510	0.000974	0.000422	0.000552	1	0.000422	0.000974	0.004857	96255.17	467.477	480107.2	4918973	51.10347
30yr-34	5	412827	295	325	620	0.001502	0.000715	0.000787	1.36	0.000525	0.001313	0.006542	95787.69	626.6388	477371.9	4438866	46.34067
35yr-39	5	340462	313	331	644	0.001892	0.000919	0.000972	1.36	0.000676	0.001648	0.008207	95161.05	781.0008	473852.8	3961494	41.62936
40yr-44	5	260845	349	379	728	0.002791	0.001338	0.001453	1.36	0.000984	0.002437	0.01211	94380.05	1142.947	469042.9	3487641	36.95316
45yr-49	5	198000	442	418	860	0.004343	0.002232	0.002111	1.36	0.001641	0.003753	0.018588	93237.11	1733.114	461852.7	3018598	32.3755
50yr-54	5	134158	488	410	898	0.006694	0.003638	0.003056	1.36	0.002675	0.005731	0.028249	91503.99	2584.891	451057.7	2556745	27.94135
55yr-59	5	109144	582	492	1,074	0.00984	0.005332	0.004508	1.36	0.003921	0.008429	0.041274	88919.1	3670.025	435420.4	2105688	23.68094
60yr-64	5	82439	811	486	1,297	0.015733	0.009838	0.005895	1.36	0.007234	0.013129	0.063558	85249.08	5418.245	412699.8	1670267	19.59279
65yr-69	5	57067	933	507	1,440	0.025233	0.016349	0.008884	1.36	0.012021	0.020905	0.099337	79830.83	7930.159	379328.8	1257567	15.7529
70yr-74	5	37251	1,059	486	1,545	0.041475	0.028429	0.013047	1.36	0.020904	0.03395	0.15647	71900.67	11250.31	331377.6	878238.6	12.21461
75yr-79	5	22758	1,116	472	1,588	0.069778	0.049038	0.02074	1.36	0.036057	0.056797	0.248675	60650.36	15082.25	265546.2	546861	9.016616
80yr-84	5	12217	1,348	464	1,812	0.127453	0.094816	0.032637	1.36	0.069718	0.102355	0.4075	45568.11	18568.99	181418.1	281314.8	6.173502
85&over	3.7	6605	1,547	561	2,108	0.244974	0.179779	0.065195	1.36	0.132191	0.197385	0.534973	26999.12	14443.8	99896.75	99896.75	3.7
total		4636463	11,008	8,844	19,852	0.004105											

Cause of Death Elimination Life Expectancy Table — Method 2

SEX/AGE	n(no.of Intervals)	Female Population	C-P Deaths	Other Deat	ToTal death	MxTotal	Mx-CP	Mx-others	RR-CP	xCPclean	xtotalclea	QxTotal	lxTotal	dxtotal	Lx	Txtotal	Extotal
<1	1	129201	528	2,020	2,548	0.019721	0.004087	0.015635	3.35	0.00122	0.016854	0.016742	100000	1674.158	98995.51	7464069	74.64059
1 YR-4YR	4	455632	437	440	877	0.001925	0.000959	0.000966	1	0.000959	0.001925	0.007676	98325.84	754.7057	391492.1	7365074	74.90476
5yr-9yr	5	478433	118	190	308	0.000644	0.000247	0.000397	1	0.000247	0.000644	0.003214	97571.14	313.5614	487071.8	6973582	71.47177
10yr-14	5	433907	110	151	261	0.000602	0.000254	0.000348	1	0.000254	0.000602	0.003003	97257.57	292.0685	485557.7	6486510	66.69414
15yr-19	5	556091	113	191	304	0.000547	0.000203	0.000343	1	0.000203	0.000547	0.00273	96965.51	264.6804	484165.8	6000952	61.88749
20yr-24	5	581667	201	229	430	0.000739	0.000346	0.000394	1	0.000346	0.000739	0.003689	96700.83	356.7733	482612.2	5516786	57.05004
25yr-29	5	523779	221	289	510	0.000974	0.000422	0.000552	1	0.000422	0.000974	0.004857	96344.05	467.9087	480550.5	5034174	52.25205
30yr-34	5	412827	295	325	620	0.001502	0.000715	0.000787	1.77	0.000404	0.001191	0.005937	95876.14	569.2356	477957.6	4553624	47.49486
35yr-39	5	340462	313	331	644	0.001892	0.000919	0.000972	1.77	0.000519	0.001492	0.00743	95306.91	708.1624	474764.1	4075666	42.76359
40yr-44	5	260845	349	379	728	0.002791	0.001338	0.001453	1.77	0.000756	0.002209	0.010984	94598.75	1039.048	470396.1	3600902	38.06501
45yr-49	5	198000	442	418	860	0.004343	0.002232	0.002111	1.77	0.001261	0.003372	0.016721	93559.7	1564.373	463887.6	3130506	33.45998
50yr-54	5	134158	488	410	898	0.006694	0.003638	0.003056	1.77	0.002055	0.005111	0.025233	91995.32	2321.363	454173.2	2666618	28.98645
55yr-59	5	109144	582	492	1,074	0.00984	0.005332	0.004508	1.77	0.003013	0.00752	0.036908	89673.96	3309.723	440095.5	2212445	24.6721
60yr-64	5	82439	811	486	1,297	0.015733	0.009838	0.005895	1.77	0.005558	0.011453	0.055672	86364.24	4808.074	419801	1772349	20.5218
65yr-69	5	57067	933	507	1,440	0.025233	0.016349	0.008884	1.77	0.009237	0.018121	0.086679	81556.17	7069.196	390107.8	1352548	16.58426

70yr-74	5	37251	1,059	486	1.545	0.041475	0.028429	0.013047	1.77	0.016061	0.029108	0.135668	74486.97	10105.49	347171.1	962440.6	12.92093
75yr-79	5	22758	1,116	472	1,588	0.069778	0.049038	0.02074	1.77	0.027705	0.048445	0.216057	64381.48	13910.08	287132.2	615269.5	9.556622
80yr-84	5	14217	1,348	464	1,812	0.127453	0.094816	0.032637	1.77	0.053568	0.086205	0.354605	50471.4	17897.4	207613.5	328137.3	6.50145
85&over	3.7	8605	1,547	561	2,108	0.244974	0.179779	0.065195	1.77	0.10157	0.166765	0.47155	32574	15360.26	120523.8	120523.8	3.7
total		4836483	11,011	8,841	19,852	0.004105											

Cause of Death Elimination Life Expectancy Table Method 3

SEX/AGE	n(no.of intervals)	Female Population	C-P Deaths	Other Deat	ToTal death	MxTotal	Mx-CP	Mx-others	RR-CP	xCPclean	xtotalclea	QxTotal	lxTotal	dxtotal	Lx	Txtotal	Extotal
<1	1	128201	528	2,020	2,548	0.019721	0.004087	0.015635	2.3	0.001777	0.017411	0.017291	100000	1729.094	98952.54	7386885	73.86885
1 YR-4YR	4	455632	437	440	877	0.001925	0.000959	0.000966	1	0.000959	0.001925	0.007676	98270.91	754.284	391273.3	7287922	74.16155
5yr-9yr	5	478433	118	190	308	0.000644	0.000247	0.000397	1	0.000247	0.000644	0.003214	97516.62	313.3862	486799.6	6896649	70.7228
10yr-14	5	433907	110	151	261	0.000602	0.000254	0.000348	1	0.000254	0.000602	0.003003	97203.24	291.9053	485286.4	6409849	65.94276
15yr-19	5	556091	113	191	304	0.000547	0.000203	0.000343	1	0.000203	0.000547	0.00273	96911.33	264.5326	483695.3	5924563	61.13385
20yr-24	5	581667	201	229	430	0.000739	0.000346	0.000394	1	0.000346	0.000739	0.003689	96646.8	356.5739	482342.6	5440668	56.29434
25yr-29	5	523779	221	289	510	0.000974	0.000422	0.000552	1	0.000422	0.000974	0.004857	96290.22	467.6473	480282	4958325	51.49355
30yr-34	5	412807	295	325	620	0.001502	0.000715	0.000787	1.48	0.000483	0.00127	0.00633	95822.58	606.5868	477596.4	4478043	46.73285
35yr-39	5	340462	313	331	644	0.001892	0.000919	0.000972	1.48	0.000621	0.001593	0.007935	95215.99	755.5681	474191	4000447	42.01444
40yr-44	5	250845	349	379	728	0.002791	0.001338	0.001453	1.48	0.000904	0.002357	0.011716	94460.42	1106.693	469535.4	3526256	37.33051
45yr-49	5	198000	442	418	860	0.004343	0.002232	0.002111	1.48	0.001508	0.003619	0.017935	93353.73	1674.29	462582.9	3056720	32.74342
50yr-54	5	134158	488	410	898	0.006694	0.003638	0.003056	1.48	0.002458	0.005514	0.027194	91679.44	2493.175	452164.3	2594137	28.29574
55yr-59	5	109144	582	492	1,074	0.00984	0.005332	0.004508	1.48	0.003503	0.008111	0.039748	89186.26	3544.971	437068.9	2141973	24.01685
60yr-64	5	82439	811	486	1,297	0.015733	0.009838	0.005895	1.48	0.006647	0.012542	0.060805	85641.29	5207.403	415188	1704904	19.9075
65yr-69	5	57067	933	507	1,440	0.025233	0.016349	0.008884	1.48	0.011047	0.019931	0.094925	80433.89	7635.216	383081.4	1289716	16.03449
70yr-74	5	37251	1,059	486	1,545	0.041475	0.028429	0.013047	1.48	0.019209	0.032255	0.149242	72798.67	10864.6	336831.9	906634.8	12.454
75yr-79	5	22758	1,116	472	1,588	0.069778	0.049038	0.02074	1.48	0.033134	0.053874	0.237395	61934.08	14702.81	272913.4	569802.9	9.200152
80yr-84	5	14217	1,348	464	1,812	0.127453	0.094816	0.032637	1.48	0.064065	0.096702	0.389376	47231.27	18390.72	190179.5	296889.6	6.285869
85&over	3.7	8605	1,547	561	2,108	0.244974	0.179779	0.065195	1.48	0.121472	0.186667	0.51338	28840.55	14806.17	106710	106710	3.7
		4836483															

ly for low, middle and high educational levels

	Qx-LSES	Qx-MSES	Qx-HSES	Px-LSES	Px-MSES	Px-HSES	lx-LSES	lx-MSES	lx-HSES	dx-LSES	dx-MSES	dx-HSES
5.64E-05	0.006658	0.004401	0.000282	0.993342	0.995599	0.999718	100,000	100,000	100,000	665.8275	440.1351	28.18682
8.2E-05	0.007548	0.005516	0.00041	0.992452	0.994484	0.99959	99334.17	99559.86	99971.81	749.7718	549.1917	41.00072
0.000103	0.009497	0.006943	0.000517	0.990503	0.993057	0.999483	98584.4	99010.67	99930.81	936.2831	687.3922	51.61594
0.000159	0.014553	0.010646	0.000793	0.985447	0.989354	0.999207	97648.12	98323.28	99879.2	1421.11	1046.745	79.24443
0.003143	0.020127	0.01612	0.015591	0.979873	0.98388	0.984409	96227.01	97276.54	99799.95	1936.716	1568.058	1556.03
0.004843	0.030849	0.024734	0.023927	0.969151	0.975266	0.976073	94290.29	95708.48	98243.92	2908.734	2367.233	2350.67
0.00712	0.045024	0.036151	0.034978	0.954976	0.963849	0.965022	91381.56	93341.25	95893.25	4114.363	3374.359	3354.154
0.011384	0.071028	0.05718	0.055344	0.928972	0.94282	0.944656	87267.19	89966.89	92539.1	6198.439	5144.316	5121.514
0.021485	0.096426	0.134077	0.101948	0.903574	0.865923	0.898052	81068.76	84822.57	87417.58	7817.132	11372.72	8912.072
0.035314	0.153721	0.211261	0.162245	0.846279	0.788739	0.837755	73251.62	73449.85	78505.51	11260.35	15517.09	12737.16
0.059412	0.245731	0.331526	0.258642	0.754269	0.668474	0.741358	61991.28	57932.76	65768.35	15233.16	19206.22	17010.45
0.108519	0.407462	0.532581	0.426803	0.592538	0.467419	0.573197	46758.12	38726.54	48757.9	19052.14	20625.04	20810.03
0.208581	0.533631	0.680929	0.556868	0.466369	0.319071	0.443132	27705.97	18101.5	27947.87	14784.78	12325.83	15563.27